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PRINCIPLES FOR IMPROVING PATIENT CARE IN THE HOSPITAL LABOR AND DELIVERY SUITE

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If one views the picture of maternity care broadly, there are two areas that are of greatest importance to the life of the expectant mother and the fetus: (1) the antepartum period and (2) the period during labor and delivery. A blueprint for antepartum care has already been briefly described.[1] This report will describe many of the essential features of care during labor and delivery that are necessary to protect the life and promote the well-being of the expectant mother, the fetus, and the newborn. These features are described as a result of surveys of the 106 hospitals in New York City having maternity and newborn services. The surveys were conducted by members of consultant survey teams of the maternity and newborn division of the New York City Department of Health.

OBSTETRIC SUPERVISION

It would be expected that the maternity service, of which the labor and delivery suite is part, should be under the direct medical supervision of a qualified obstetrician. Such an obstetric director should be responsible for the establishment of the technical obstetric policies and procedures for the care of patients during labor and delivery. Daily rounds would be made by the director, or by an obstetrician or staff of obstetricians, to provide the necessary supervision so that the established policies and procedures would be well carried out.

Of major import to the expectant mother and her fetus are policies regarding the indications that warrant consultation with a qualified obstetrician. Such consultation indications usually include the following: major obstetric operative procedures; forceps delivery other than outlet forceps; serious obstetric or medical complications, such as toxemia, bleeding, cardiac disease, premature labor, diabetes, and the like; breech presentation, and labor of more than 24 hr. in primiparous patients. The question of the need of consultation for multiparous patients with breech presentation is in the process of being studied.

In addition to adequate obstetric supervision at the attending staff level, there should be adequate obstetric house staff coverage, which serves two functions: (1) the training of interns and residents in obstetrics and (2) the provision of continuous medical supervision of the patient during labor and delivery. Ideally, every maternity service should be of such caliber as to be approved for the training of interns and residents in the specialty and therefore should be of the type that will ensure eligibility for the American Board of Obstetrics and Gynecology for house staff members who participate in the full residency system of training. Since the great bulk of obstetrics throughout the country is performed by physicians in general practice, there should also be adequate opportunity for the training of these future practitioners. Delineation of functions of the attending, resident, and intern staff is necessary if there is to be adequate protection for the patient in labor and during delivery.

It is desirable that the physicians who give antepartum care to patients in the hospital's antepartum clinic be members of the hospital's inpatient staff so that there will be continuity of medical care of the patient.

ANESTHESIA SERVICE

It is a curious dichotomy in the pattern of medical practice that there is a discrepancy in the quality of anesthesia service for the surgical operating room and for the delivery suite. In these hospitals that have well-organized anesthesia departments, it is not uncommon to find anesthesia service being rendered to surgical patients by well-trained and qualified physician and nurse anesthetists; in contrast, the anesthesia service for

From the Maternity and Newborn Division, New York City Department of Health.

A qualified obstetrician is herein described as one who holds any one or combination of the following ranks: (1) specialist certified by the American Board of Obstetrics and Gynecology, (2) fellow of the American College of Surgeons (in the specialty of obstetrics and gynecology), and (3) associate attending obstetrician or higher at a voluntary or municipal hospital.

1. Gold, E. M.; Wallace, H. M., and Losty, M. A.: A Blueprint for Changing Concepts of Antepartum Care, Am. J. Pub. Health **40**: 792-797 (July) 1950.

the delivery suite is usually of a "pick-up" nature—anyone may render such anesthesia. This survey finding is reflected in the fact that in New York City anesthesia has now emerged as one of the major causes of maternal mortality, being outranked only by hemorrhage, infection, heart disease, and toxemia.[2] This dichotomy becomes even more marked when it is realized that there are usually at least two lives at risk in the delivery room (mother and fetus), while only one life is at risk in the surgical operating room.

It follows then that each hospital having maternity service should have available at all times a well-organized anesthesia department, part of whose functions is the provision of anesthesia service to those obstetric patients who are to receive it. Either well-trained and qualified physician or nurse anesthetists may be used. Such personnel will also play a major role in the determination of feeding policies during labor and of resuscitation of the newborn.

NURSING STAFF

It is generally agreed that the nursing care of the patient in labor and during delivery is one of the strategic areas of patient care in any hospital. Just as there is a qualified obstetrician responsible for the medical supervision of patients in labor and delivery, so too there should be a well-qualified nurse in charge of the labor and delivery suite. Such a member of the nursing staff should have had advanced preparation in the field of maternity nursing and should be directly responsible to the nursing supervisor of the maternity service. There should be a staff of registered professional nurses for each tour of duty in sufficient numbers to provide or supervise good patient care. The number will depend on the size of the maternity service and the number of deliveries to be done.

There have been no studies undertaken to indicate what sized nursing staff is necessary for patients in labor and during delivery nor any studies to indicate the proper proportion between the nursing staff at the registered professional nurse level and the nursing staff at the level of practical nurses, aides, attendants, and the like. It is, however, generally agreed that labor patients, particularly those who receive analgesia, should not at any time be left unattended. It is likewise agreed that there should be one member of the nursing staff, and preferably two, functioning in the delivery room during a delivery. If the labor and delivery suite has a recovery room, an adequate nursing staff must be provided in this area if the purpose for which the recovery room is intended is to be served.[3]

The personnel of the labor and delivery suite will depend to a certain extent on whether there is a central supply room in the hospital, or whether the making and sterilization of supplies for labor and delivery are done in the delivery room suite itself. The former arrangement is more efficient and economical. The latter requires additional nursing supervisory and auxiliary personnel, as well as space in the labor and delivery suite, and furthermore requires so careful a definition of duties in this area, that the time of the registered professional nurse, who is essential for good patient care, is not spent in washing gloves, making supplies, and the like. Such duties constitute a waste of skill and training.

PHYSICAL SETUP

The physical setup of a labor and delivery suite may be functionally divided into the following areas: (1) areas of patient care, including admission room, labor room, delivery rooms, and recovery room; (2) areas for staff functions, including physicians' dressing room, nurses' dressing room, office of the obstetric director, office of the nurse in charge, office for the anesthetist, chart room or area, and scrub-up, and (3) accessory facilities, including obstetric cleanup, nurses' workroom, sterilization room (if a central supply room is not provided), laboratory, storage, and utility room.

AREAS OF PATIENT CARE

Admission Room.—It is essential that the labor unit contain an admission room, where the new labor patient may be received into the unit, and where the admission history and physical examination may be performed. Here too the patient may be bathed and prepared for labor and delivery.

Labor Rooms.—The number of labor rooms and delivery rooms will depend on the number of maternity reservations made for a given period and on the amount of patient turnover in the maternity service. A practical index to use is the provision of one labor bed for each 10 postpartum beds. In order to provide patient privacy, each labor patient should be in a separate room. Each single room should have a minimum of 80 sq. ft. of floor space and have hot and cold running water equipped with arm, foot, or knee control, sphygmomanometer, stethoscope and the like. All equipment for the patient, such as a thermometer and bedpan, should be at the bedside. All labor beds should be equipped with sideboards to protect the patient.

Delivery Rooms.—A practical index to use in the planning of delivery rooms is one delivery room for every 20 postpartum beds. A minimum of two delivery rooms is essential in any service, for there are occasions when it is necessary to conduct more than one delivery at a time. At no time should there be two deliveries conducted in the same room at the same time. Each delivery room should be provided with adequate supplies and equipment for the protection of the life of the mother and her newborn infant, including an anesthesia machine, delivery instruments, medications, a cutdown set, a supply of plasma and intravenous fluids, a modern thermostatically controlled incubator or heated crib that should be kept warm at all times, resuscitation equipment for the newborn infant, identification equipment, and oxygen. Obviously, there must be personnel at each delivery well versed in the art of resuscitation of the newborn infant. It is desirable that a small amount of blood, both Rh positive and Rh negative of all four types, be kept in the delivery suite for emergency use, since

2. Gold, E. M., and Wallace, H. M.: A Study of Maternal Deaths in New York City for 1947, New York State J. Med. 49: 1676-1680 (July 15) 1949.

3. McLean, L. F.; McDowell, H. C., and Sadugor, M. S.: The Use of the Recovery Room in Lowering Meternal Mortality, New York State J. Med. 48: 1368-1370 (June 15) 1948.

hemorrhage is the major cause of maternal mortality in New York City at present. Adequate lighting is essential, as well as provisions for the prevention of electrostatic hazards.[4] Many hospital authorities consider it desirable to soundproof and air-condition the labor rooms and the delivery rooms. If cesarean sections are to be performed in the delivery suite, one delivery room should be completely equipped as an operating room. If, on the other hand, cesarean sections are performed in an operating room, the operating room should be equipped to receive and give immediate care to the newborn infant.

Recovery Room.—If there is sufficient nursing staff available, a recovery room is an additional safeguard for the patient in order to observe and evaluate her condition more fully before her transfer to the postpartum service.

Areas for Staff Functions.—Such areas should include, near the entrance to the labor and delivery unit, separate facilities where physicians and nurses can change their clothes; in addition, shower and toilet facilities for each group are necessary. Offices should be provided for the obstetric director, the nurse in charge, and the anesthetist. A chart room or area is essential if there is to be adequate recording of the patient's progress in labor and during delivery. It is obvious that adequate scrub-up facilities are necessary, preferably with direct vision into the delivery rooms.

Accessory Facilities.—Accessory facilities should include an obstetric cleanup room, where used supplies and equipment may be taken after a delivery and immediate cleaning performed. If a central supply room is not provided in the hospital, the labor and delivery unit should have its own workroom and sterilization room. Storage areas are essential. The labor unit should have a utility room, equipped to perform sterilization of bedpans, enema equipment, and the like. It is desirable that the labor and delivery unit have a small laboratory equipped to perform an admission urinalysis, emergency blood typing, cross-matching, and Rh determination.

Other Necessary Hospital Services.—As is true in the care of all hospital patients, certain other hospital services are necessary for the care of maternity patients and newborn infants. For example, the hospital should be expected to provide a general laboratory department under the immediate direction of a qualified pathologist and with an adequate and trained laboratory technician staff at all times. Such a laboratory should be able to perform bacteriological, biochemical, hemotological, and pathological determinations.

Another important general hospital service that is necessary is the x-ray department under the immediate direction of a qualified roentgenologist and with an adequate and trained x-ray technician staff at all times. Such a department should be able to perform x-ray pelvimetry according to one of the accepted techniques.

RECORDS

Adequate records of labor and delivery are important if the obstetric director is to be able to evaluate patient care. A continuous and current labor and delivery record book is essential. This record book should contain basic information regarding the identification of the patient, parity, length of labor, type of delivery, type of anesthesia, name of physician performing the delivery and name of consultant if any, name of anesthetist, and condition of fetus or newborn infant, including birth weight. Such data should be summarized monthly and then annually.

Each patient's record should contain information regarding her antepartum care; course during labor, delivery, and the period immediately after delivery; laboratory data, and consultation notes. If the patient attended the hospital's antepartum clinic, her clinic record should be available immediately on her entrance to the labor unit. If she is a private patient, her physician should be expected to send a résumé of her antepartum care to the hospital's labor and delivery unit shortly before the expected date of confinement.

The record of the newborn infant should be initially filled out in the delivery room and should include the essential data regarding the mother as well as initial condition in the delivery room.

STATISTICAL EVALUATION AND STAFF EDUCATION

If improvement in patient care is to be made, there should be opportunity for periodic review of current care. One method by which this objective may be accomplished is the institution of periodic joint staff meetings of the obstetric, pediatric, pathology, and nursing staffs, to discuss problems within the service and to review maternal, fetal, and neonatal mortality and morbidity. This system should be of great educational value to all personnel concerned with the care of maternity patients and newborn infants.

SUMMARY

This report describes some of the essential features of a labor and delivery suite of a hospital maternity service, suggested by members of a hospital consultation service, as a result of surveys of hospitals in a large metropolitan area. The suggestions include obstetric supervision, anesthesia service, nursing supervision and staff, physical setup and equipment, other necessary hospital services, records, statistical evaluation, and staff education.

125 Worth St.

4. Recommended Safe Practice For Hospital Operating Rooms, National Fire Protection Association, Boston, 1949.

Infant Feeding.—Nature produced breast milk in the mother to feed the infant for the greater part of the first year. The first teeth do not erupt before six or seven months because nature did not anticipate any need for them. Ptyalin does not appear in the saliva in appreciable amount before the third or fourth month of life. The infant does not learn to use his tongue and jaws to attempt to chew and mix and swallow solid foods before he is three and one-half or four months of age. The intestinal tract is unable to filter out large protein molecules from the food before the child is six or eight months of age. . . . There have been an endless number of various food modifications for infant feeding. None has ever surpassed or ever equaled breast milk as produced by nature. The best substitute for breast milk is a properly prepared cow's milk modified to the approximate composition of human breast milk.—M. G. Peterman, M.D., So-Called "Progress" in Infant Feeding, *Rocky Mountain Medical Journal*, June, 1951.

PRINCIPLES AND FACILITIES FOR THE CARE OF NEWBORN INFANTS IN THE HOSPITAL

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In this postwar era of hospital planning, construction and reconstruction, considerable thought and money have gone into the various hospital services. Nationally, interest in the standards of newborn care has been expressed by several groups,[1] and has, of course, been stimulated by the federal Hospital Survey and Construction Act. The pediatrician must actively participate in planning the newborn service.

PEDIATRIC CARE

The service for newborn infants should be under the direct supervision of the pediatric staff of the hospital; this principle has been adopted in many of the teaching hospitals in this country and should be extended to all hospitals. As a corollary of this principle, a qualified pediatrician, usually the director of the pediatric service, should be the director of the newborn service. He should have the responsibility for the establishment of the technical pediatric policies and procedures and for the supervision of the newborn service, so that such policies and procedures are carried out. Daily rounds on this service should be made by the director or by a staff of one or more attending pediatricians appointed by the director.

There should be pediatric house-staff coverage throughout the entire 24-hr. period. The pediatric house staff assigned to the newborn service should have no duties, during such assignment, in the care of sick infants and children on the general pediatric service but it might simultaneously be assigned to the premature center, to the well baby clinic and to the special premature follow-up clinic, if any or all of these services are provided in the hospital. House-staff duties during this period should include assignment to the delivery room for the immediate care of the newborn infant, and possibly also to the antepartum clinic of the hospital, since in these areas the pediatrician can play an important role in patient care. Patterns established in the antepartum and immediate postpartum periods play a vital role in the development of proper parent-child relationships.

The continuous review of care already given infants is an important educational tool in the improvement of such care. One form of educational activity is the periodic pediatric staff meeting, held jointly with members of the obstetric, pathologic and nursing staffs, to review mortality and morbidity. Such a plan necessitates inclusion of one or more members of the pathology department who are interested and trained in the field of neonatal pathology. It also provides for keeping current statistics for the service, including total deliveries, live births,—under and over 5½ lb. (2.5 kg.)—deaths by causes, and infants alive at the time of discharge. Such analysis might be carried further to include a breakdown by color and by type of patient, whether on private or general service.

There is, too, a responsibility for assisting parents in making plans for the continuing medical supervision of the infant after discharge from the hospital, whether such care is to be given by a private physician, hospital well baby clinic or by a community well baby clinic.

NURSING STAFF

The service for the newborn should be under the direct supervision of a registered professional nurse [2] who has had advanced training in all phases of newborn care. She should continue the training of the various persons comprising the nursing and auxiliary staff who care for newborn infants. In addition, experience has shown that there is a close parallelism between the provision of a capable, well-trained nursing supervisor and the quality of patient care rendered on the newborn service. To provide supervision of patient care over the 24-hr. period, there should also be on each shift a head nurse with advanced training in this special field.

For the direct care of the newborn there should be one nurse for every eight full-term newborn infants at all times. Observations have led us to believe that there is little or no significant difference in the amount of nursing care needed by newborn infants during the day, evening, and night periods. Members of the auxiliary nursing staff should at all times work under the supervision of the supervisor or head nurse.

Ideally, the nursing staff for newborn infants in the suspect, or observation, nursery should be separate from that caring for well newborn infants. Since this is not practical in most hospitals, if technique is carefully observed these infants can be cared for by nurses of the regular nursery service. This plan has proved satisfactory when the observation nursery is adjacent to the regular nursery.

Members of the nursing staff of the isolation nursery must be separate from those caring for well newborn infants. This principle must be scrupulously adhered to in order to avoid cross-infection between the isolation and clean nurseries.

PERSONNEL HEALTH POLICIES

The hospital should establish a plan for the health services of its personnel, including those pediatric, nursing, housekeeping and cleaning personnel working in the newborn service. Such a plan should include a preemployment and periodic, usually annual, physical examination including a chest x-ray, and a plan for the immedi-

From the Department of Pediatrics, New York Medical College, Flower and Fifth Avenue Hospitals.

1. (*a*) Standards and Recommendations for Hospital Care of Newborn Infants: Full-Term and Premature, revised edition, Evanston, Ill., American Academy of Pediatrics, 1948. (*b*) Standards and Recommendations for Hospital Care of Newborn Infants: Full-Term and Premature, Publication 292, Children's Bureau, 1943.

2. Losty, M.: Personal communication to the authors.

ate reporting of any illness suggestive of a communicable skin, gastrointestinal or respiratory disease. Such an illness would immediately exclude affected personnel from the newborn service, and they should not be permitted to return to their regular assignments until a physician has certified their recovery. The effectiveness of such a plan would depend on the early reporting of illness and early exclusion from duty. The use of routine stool, nose or throat bacteriological cultures has proved of doubtful value in the control of infection in a service.

PHYSICAL LAYOUT OF NURSERIES

The ideal physical layout consists of the following functional areas: (1) clean, or regular, nurseries for full-term newborn infants; (2) the suspect, or observation, nursery; (3) the isolation nursery, and (4) the formula-preparation room.

Clean, or Regular, Nurseries.—Functionally, the most practical location of the clean, or regular, nurseries is in close proximity to the respective postpartum rooms. This location will facilitate the easy transportation of all normal full-term newborn infants to their mothers for feeding.

Ideally, small nurseries should be provided. The most efficacious plan is to provide space for only eight full-term newborn infants in each nursery room. The chief advantage of such a provision is that, if infection should occur in a specific nursery, its spread, if any, would be limited to only a small number of infants.

A minimum average of 30 sq. ft. per bassinet should be provided in each nursery room to give adequate space around each bassinet, for the completely individualized care of each infant. This average square footage will provide nursery space for a sink and diaper, linen and disposal cans and will make it relatively easy to wheel the scale to the cribside for weighing. In addition, it will accommodate chairs in which the nurse can sit and hold those babies who for one reason or another cannot be taken to their mothers for feeding.

Equipment for each nursery room should be the simplest possible. There should be individual bassinets; a weighing scale on a movable table; a sink providing hot and cold running water and equipped with arm, foot or knee control; bar soap; disposable towels, and discard cans operated by a foot pedal for linen, diapers, and other soiled materials. Each bassinet should have a compartment for a 24-hr. supply of all equipment needed for each infant's care—diapers, linen, oil and its container, and a thermometer. The purpose of this principle of individual care is again to minimize the possibility of cross-infection. There should also be one modern, efficient incubator for every 16 infants, and oxygen and suction equipment for each nursery.

Each nursery room or set of two nursery rooms should have an anteroom and an examination and treatment room. The anteroom will be used primarily as a chart area and will also be the area where the physician scrubs and dons a mask and gown prior to entering the examination and treatment room. The examination and treatment room, as the name implies, is to be used for examination and treatment of infants and also for operative circumcision; it therefore should contain supplies and equipment for this purpose. In addition, it should contain a bottle warmer and refrigerator for formulas. As much glass panelling as possible should be provided between this accessory work space and the nursery proper for ease of observation and supervision.

The Suspect, or Observation, Nurseries.—When the regular nurseries for newborn are located on more than one floor, a suspect, or observation, nursery should also be provided on each floor to lessen the possibility of the introduction of infection from one floor to another. The suspect and clean nurseries should be as close to each other as possible, because they will possibly both be staffed by the same nurse. The data presented above on the regular nurseries, regarding capacity, size and equipment apply equally well to the suspect nursery. The suspect nursery should contain one bassinet for every eight bassinets in the clean, or regular, nurseries.

The suspect nursery is for observation of any newborn infant suspected of having an infectious condition. This nursery, therefore, should also care for newborn infants after a ritual circumcision because of their exposure to fairly large numbers of outside visitors and because of possible infection resulting from such exposure. The suspect nursery should be looked upon as a hub for diagnosis. Thus, any newborn infant suspected of having an infectious condition should be immediately removed to the suspect nursery. If, after a brief period of observation, a diagnosis of infection is made, he is to be removed to the isolation nursery; if on the other hand, an infant is found not to have an infectious condition, he may be returned to the regular nursery.

The Isolation Nursery.—When a general hospital has a well-organized pediatric service, it is more desirable to locate the isolation nursery on this service, since this service is better geared to the care of sick infants than the clean newborn service usually is. Furthermore, it is desirable that infection be removed from the clean service as quickly as possible. Once an infant has been removed to the isolation nursery because of the diagnosis of an infectious condition, he cannot be allowed to return to the regular, or clean nurseries. The data presented above on the regular nurseries regarding capacity, size, and equipment apply equally well to the isolation nursery. The isolation nursery should have one bassinet for each eight bassinets in the clean, regular nursery.

Formula-Preparation Room.—In this era when artificial feeding of newborn infants is not uncommon, formula preparation has become an important function of the newborn service. The formula room should be located away from any infected or contaminated area of the hospital and should be near, or easily accessible to, the largest number of infants it may serve.

In order to separate clean and unclean activities, it is desirable to provide a reception and clean-up room, and a formula-preparation room. In the former room, bottles, nipples, and nipple caps are received from the nurseries and thoroughly washed. In the latter room, formula mixtures are prepared and poured into formula bottles, which are nippled and nipple capped; terminal sterilization is performed at 230 F. for 10 min. or 212 F. for 25 min. Refrigeration may be provided centrally within the formula room or locally within the work-space area of the nurseries. Bacteriological testing of formulas and

nipples should be performed by the hospital periodically, preferably weekly, to assure a safe product for the infants.

In general, the recommendations of the American Hospital Association manual on procedures and layout for the infant formula room [3] are valuable and serve as an excellent guide in formula preparation.

POLICIES AND PROCEDURES

The pediatric director, with the help of the supervising nurse of the newborn service, should establish policies and procedures for the functioning of this service. Such policies and procedures should be thoroughly discussed with all groups concerned prior to their establishment, so that those finally agreed upon will be feasible as well as scientifically desirable. For example, some of the procedures with which both the pediatric and nursing care are concerned and in which the availability of nursing time is a factor are (1) frequency of weighing of normal full-term infants; (2) daily care of the skin; (3) frequency of routine temperature taking; (4) use of routine house formulas; (5) use of abdominal binders and umbilical cord dressings; (6) use of gowns and masks by nursing personnel constantly working in clean nurseries.

In such procedures, emphasis must be placed on the elimination of nonessentials; thus, the time of the available nursing staff will be conserved for the really necessary jobs. The available time then may be used for essential procedures and for taking the normal full-term babies to their mothers for all daytime feedings. This latter practice eliminates the universally prevalent technique of propping bottles, with its potential danger of aspiration of formula; at the same time it gives the mother an opportunity to learn how to hold and feed her baby during the period of hospital stay and establishes the normal emotional child-mother relationship. Nursing time should also be available for teaching and educating the mother and father so that they will approach care of the infant with confidence and security.

In the establishment of such policies and procedures, it is usually desirable that a manual be prepared for joint use of the medical, nursing, and auxiliary staffs. This manual should contain emergency standing orders as well as a listing of the agreed-upon policies and procedures. One item of considerable importance is the establishment of policies for consultation with a qualified pediatrician, i. e., for sick and potentially sick newborn infants and for premature infants. In addition, similar policies should be established for consultation with the respective specialists for such conditions as congenital, orthopedic and cardiac deformities, and surgical conditions.

RECORDS

Complete and detailed records of the infant's hospital experience are as necessary for newborn infants as for all hospital patients. The newborn infant should have a record of his own that is separate from that of his mother. However, his record should include pertinent data regarding the mother's course during the antepartum, labor and delivery periods. Such data might include complications of pregnancy, labor and delivery; serological test and determination of the Rh factor; analgesia; anesthesia; length of labor and type of delivery. The record of the newborn infant should begin with his condition and care in the delivery room and should continue with his hospital neonatal course. It should include such items as weight, temperature, respiration, feeding and reactions to feeding, and stools. Admission and discharge physical examinations, progress notes, consultation notes and laboratory data, when indicated, are a necessity.

In addition to the clinical record of the individual newborn infant, it is desirable to have a worksheet or workbook in each nursery kept on a daily basis. Such a book or sheet should contain the name of each infant and such essential information as age; weight; temperature; condition of eyes, mouth, cord, skin, and buttocks; number, size, color and consistency of stools (a code may be used); urine; type and amount of feedings; treatment; medications, and remarks.

COORDINATION OF CARE IN THE HOSPITAL AND IN THE HOME

Ideally, a hospital should have a plan by which, at the request of a physician, all newborn infants are visited in the home by members of a community nursing agency immediately after discharge from the hospital. The purpose of such home visiting is to help the parents carry out the instructions on care of the baby in the home; in other words, such visiting is a link between the teaching program within the hospital and that given through health supervision during the infant's early months and years of life.

However, in this era of nursing shortages, it may not be possible to have all newborn infants visited in the home. Therefore, priority for visiting must be established. Such priority may be given the following infants: those prematurely born; the newborn with birth injuries or congenital malformations, and those born of primiparous or insecure mothers. Each of the above categories may have special problems with which the parent or parents may need considerable assistance.

ACCESSORY SERVICES NECESSARY

Laboratory and x-ray services with an adequate technical staff should be provided within the hospital, so that they will be available when needed for the newborn. There should be a qualified hematologist or pathologist and radiologist, respectively, in charge of such services. Furthermore, post-mortem facilities should be provided, if there is to be an opportunity of learning the lessons potentially available from a review of individual neonatal deaths.

Social service is most important. A social worker can be of great assistance in such problems as placement of illegitimate infants and of babies with such deformities as mongolism, hydrocephalus, and meningocele. She can assist in making a plan for further care of infants with birth injury, congenital cardiac, orthopedic and ophthalmological deformities.

CONCEPT OF FUNCTION OF THE NEWBORN SERVICE

In a general hospital the maternity and newborn services are the only two largely composed of well or normal people. Despite this unique difference, these normal, well, maternity patients and newborn infants have been

3. Procedures and Layout for Infant Formula Room, Chicago, American Hospital Association, 1949.

treated and managed as sick individuals. To our way of thinking, this treatment is entirely artificial and unnecessary and one which works to the disadvantage of the patient. For example, it seems useless to take the temperature of a normal full-term newborn infant routinely every four hours. It likewise seems impractical to look upon such an infant as a "sterile product" that must remain such and therefore cannot be taken out to its mother until the time of discharge of mother and baby from the hospital.

More and more within the past few years the obstetrician, pediatrician and general practitioner have come to recognize the fallacies in such practices. Certainly, rooming-in when carefully planned has several advantages for the parents and their newborn infant. Not only can the postpartum hospital stay be used as a period of instruction of the parents, but, even more important, the rooming-in plan is an excellent device to foster desirable parent-child relationships and the development of a healthy personality in the child.

SUMMARY

A plan for hospital service for newborn infants is discussed. Topics of this discussion are pediatric care, an adequate nursing staff, personnel health policies, the physical layout and equipment of nurseries, policies and procedures, records, coordination of care in the hospital and in the home, and accessory services. The value of a more flexible system in the hospital care of newborn infants is pointed out.

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MIDFORCEPS OPERATIONS

INDICATIONS FOR AND RESULTS OF ONE THOUSAND CASES BASED ON PELVIRADIOGRAPHY AND PROGRESS OF LABOR

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In modern obstetrics, the midforceps operation has become the chief delivery room problem. This operation is usually selected for completion of deliveries in cases of midpelvic arrest caused by malposition or poor uterine or abdominal contractions.

The definition of a type of forceps operation must include reference to fetal and pelvic planes that are ascertainable and constant. For the fetal point of reference, the use of the biparietal diameter is not wise, because in the average obstetrical forceps operation, the obstetrician contents himself with the station of the most dependent portion of the presenting part and the location of the sutures and fontanels for determining position and station. The location of the biparietal diameter is easily determined by a lateral roentgenogram, but determination of its level in the pelvic axis by manual examination is difficult. I, therefore, use and urge the adaptation of the most dependent portion of the presenting part, excluding a caput succedaneum.

Likewise, the pelvic points of reference must be fixed and locatable either by roentgenogram or manually. The use of the ischial spines for designation of the midpelvis and the perineum for the outlet is illogical, because the perineum may be absent in third degree tears or depressed in lacerated perineums in relation to the true pelvis, and furthermore the muscles are not discernible by roentgenograms. The logical plane for one to select as the station of the outlet is the level of the ischial tuberosities, which are fixed and easily located roentgenographically or manually. The plane of the inlet is, of course, the linea terminalis at the level of or slightly below the true conjugate. Therefore, the following definitions are suggested.

A midforceps operation is an obstetrical operation by which a fetus which presents cephalically and whose presenting part is located in the midpelvis, that is, between the planes of the ischial spines and ischial tuberosities, is delivered by forceps. In a floating forceps delivery, the most dependent part lies above the inlet. In a high forceps operation, the presenting part lies between the inlet and spines, and in a low forceps, it lies below the tuberosities. An outlet forceps operation is delivery of a fetus by forceps whose presenting part is easily visible and is distending the perineum and vulva.

Midforceps deliveries are sometimes performed when the patient has been fully dilated for two hours without progress, and the cause of the delay is not known. In some cases, a midforceps operation is indicated when undue maternal distress is apparent either from hemorrhage, toxemia, cardiac disease, tuberculosis, infection, or exhaustion from prolonged labor.

In cases of sudden development of fetal distress from a prolapsed cord, intrapartum hemorrhage, or intrauterine asphyxia from the labor itself, if proper conditions exist, a midforceps operation is selected as the best chance for delivery of a baby before it succumbs to the unfavorable influences of its environment.

In some cases of midpelvic arrest caused by cephalopelvic disproportion, the forceps are wrongly used to terminate labor, and the maternal morbidity and fetal mortality are usually prominent in this type of case. Whereas forceps are contraindicated in cases of disproportion, they are sometimes of great value in delivery of the arrested vertex in cases of pelvic contraction. If the planes and segments of contraction and the pelvic architecture are determined by pelviradiography, the forceps may be used to direct the vertex into relatively more ample planes and segments of the pelvis. By control of the position of the vertex, the various cephalic

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diameters may be presented to the diameters of the pelvis in such a manner that delivery is facilitated. It is by such considerations that Mengert [1] and Caldwell, Moloy, and D'Esopo [2] have attempted to make the forceps operation more of an art than a simple trial and error method of the application of force to a problem arising from the mechanism of labor.

In recent years the indications for forceps operations have been extended to include the solution of soft part dystocia in many of its aspects. In the first stage of labor the cervix may be dilated to 8 cm. and then, notwithstanding the proper combination of time, rest, sedation, intravenous glucose solution, and oxytoxic stimulation, dilatation may cease. Maternal or fetal distress at this stage may indicate the advisability of the termination of labor. If all the other conditions for a forceps operation are present except the fully dilated cervix, namely, engaged head, no disproportion, membranes ruptured, and position known, one may occasionally be able to apply forceps and deliver successfully if the cervix is negotiable or can be made so by regional pudendal, caudal, or saddle block anesthesia, or by general anesthesia. There are also some cases of constriction ring dystocia that can be handled in a similar manner.

Dystocia at the inlet is more readily recognized both clinically and by roentgenogram and early enough for abdominal delivery to be selected rather than a high forceps operation. Outlet dystocia alone must be very rare, because the outlet is almost always more ample than the midpelvis. The only exception is if the pelvis is deformed by injury or metabolic or deficiency diseases, in which case significant outlet dystocia without midpelvic dystocia is possible.

In my own experience, therefore, the chief problem in the delivery room is midpelvic arrest. I and many of my colleagues and associates give more consideration to the decision of performing a midforceps operation than we do to any other obstetrical problem. We look for indications and contraindications to justify our decision. If the result is poor, a question arises: Did we interfere too soon or too late? I know of no other obstetrical decision about which there is always so much criticism and difference of opinion. I have seen many well-trained obstetricians hesitate and procrastinate before attempting an inevitable midforceps operation. On the other hand, we have undoubtedly at times interfered too soon with tragic results. In an attempt to more clearly define the indications and contraindications for the operation, a study of 1,000 patients delivered by midforceps was made. To eliminate unknown factors relating to pelvic architecture and size, only patients who had been subject to pelviradiography were included.

1. Mengert, W. F., and Eller, W. C.: Am. J. Obst. & Gynec. **52**: 1032, 1946. Eller, W. C., and Mengert, W. F.: Am. J. Obst. & Gynec. **53**: 252, 1947.

2. Caldwell, W. E., and Moloy, H. C.: Am. J. Obst. & Gynec. **26**: 479, 1933. Caldwell, W. C.; Moloy, H. C., and D'Esopo, D. A.: ibid. **28**: 482, 1934; ibid. **28**: 824, 1934; ibid. **30**: 763, 1935; ibid. **32**: 727, 1936; ibid. **36**: 928, 1939.

3. Bradford, W. Z.; Woltz, J. H. E., and Bradford, W. B.: Am. J. Obst. & Gynec. **58**: 1093, 1949.

INCIDENCE

The incidence of midforceps operations as represented in this series is difficult to determine, because the material analysed is drawn from many different sources. All were patients on whom midforceps operations were performed, who had had pelviradiography done, and at whose delivery I was present either as the attending or consulting obstetrician or the radiologist. The patients were delivered at the following institutions: Queens General, St. Joseph, Rockaway Beach, Beth Israel, South Nassau Communities, and Mercy Hospitals, all in New York State. In my own practice, the incidence was 11%.

Of the midforceps operations, 72.8% were done on primiparas and 27.2% on multiparas. Since the distribution of primiparas and multiparas in this area is about equal it is clear that the operation is definitely more frequent in primiparas. However, since 27.2% of the midforceps operations were performed on multiparas, it follows that when a multipara is in labor the entire problem is not just for one to await a call that the patient is ready, and then to arrive in time to deliver the baby.

DURATION OF LABOR

The duration of labor at times is difficult to estimate because the onset is often indefinite. A prolonged labor is by definition a labor of 30 hours. From the viewpoint of the patient and her family, labor usually causes anxiety from the time of admission to the hospital. In this series, labor was considered as beginning when contractions were regular, were under 10 minutes, and affected the cervix either by effacement or dilatation. The irregular contractions of uterine hyperirritability were not considered as part of labor.

TABLE 1.—*Incidence of Normal and Prolonged Labor*

Stage of Labor	Normal, %	Prolonged, %
First stage	81.8	18.2
Second stage	91.4	8.6
Both stages	....	2.4

The first and second stages of labor are somewhat different from each other both in the muscles utilized and in the physiological result. The difference is emphasized by study of the incidence of prolonged labor of each stage. I consider a prolonged first stage one over 24 hours. Table 1 indicates that even though a prolonged first or second stage occurred in 26.8% of the cases, in only 2.4% were they combined. This further emphasizes the essential difference between the two stages. In a series of midforceps deliveries, a fairly high incidence of prolonged labor is expected. The usual incidence of prolonged labor is about 3% of all cases. In a series of 85 prolonged labors, Bradford and Woltz [3] reported an incidence of midforceps of 49% and 11% of other major operative procedures. The midforceps operation has always been a mainstay in the termination of prolonged labor, but it should be realized that after 50 hours of labor, 60% of the cases never reach full dilatation, and the conclusion is inescapable that more labor will seldom result in further dilatation or easier delivery. After prolonged labor, the fetal mortality and maternal

morbidity of midforceps operations rise sharply, and the attempt to shorten labor before the 50 hour deadline by safe means should be considered.

INDICATIONS

The indications for forceps operations have been divided according to custom into maternal and fetal indications (table 2). If more than one indication was present, for the sake of simplicity only the major indication was considered. However, in 5.6% of the patients, no obstetrical indication was involved. These operations were usually performed because the station of the vertex was misjudged to be lower than it actually was. Others were delivered because they were under anesthesia when the diagnosis was made. These were usually in multiparas who were anesthetized prematurely by the nursing

TABLE 2.—*Maternal and Fetal Indications for Forceps Operations*

Indication	Percentage
No indication	5.6
Maternal	
Arrest	91.8
Poor contractions	21.8
Lack of voluntary effort	11.8
Combination of poor contractions and lack of voluntary effort	6.0
Pelvic soft part dystocia	8.0
Contracted pelvis	2.0
Disproportion	0.4
Impacted shoulder	0.4
Imprisoned anterior lip	0.2
Cervical rigidity	0.2
Extended head	0.2
Arrest occiput posterior	5.2
Persistent malposition	24.6
Malposition	11.0
Distress	0.6
Exhaustion	0
Hemorrhage	0
Tuberculosis	0
Cardiac	0.2
Toxemia	0.4
Fetal	
Distress	2.0
Asphyxia	1.6
Hemorrhage	0.2
Prolapse of the cord	0.2

staff to prevent precipitate delivery. There were some patients delivered without any other indication except that an uncooperative patient was raising such havoc or making such a loud noise that for everyone's comfort the delivery was anticipated by a midforceps operation if the conditions were favorable and no contraindications were present.

There are two groups of maternal indications: arrest or distress during labor. By arrest is meant a lack of progress in either descent or rotation. Definite progress is expected within two hours of good second stage labor contractions if the membranes are ruptured. Very slight progress is of no avail, and unless the labor is progressing toward an easy delivery, further procrastination is usually of no avail, and if no disproportion exists, the results of a midforceps delivery at this time are as good if not better than those attempted after a much longer second stage during which the baby may be damaged by cerebral concussion, intracranial hemorrhage, or be asphyxiated by a tight cord or one shortened by coils around the fetal neck. Such events cannot always be detected by auscultation of the fetal heart, and obstetricians are sometimes surprised to see an asphyxiated baby emerge when the fetal heart was regular and of a normal rate.

If the second stage contractions are weak, progress may well be unexpected within the prescribed time limit. Instead of waiting longer, after one hour I prefer to stimulate the second stage contractions by rupturing the membranes if they are still intact. This usually results in an acceleration of progress in labor. If after one-half hour the contractions are still not powerful, I use oxytocin injection (pitocin®) hypodermically in doses of 1 or 2 minims (0.06 or 0.12 cc.) every 20 minutes for one hour or an intravenous infusion of dilute oxytocin injection. If the contractions fail to result in descent and in rotation, the time indication for midforceps delivery is invoked. I usually look for progress in descent rather than in rotation, because in observing malposition by pelviradiography, I found that if rotation fails to occur during the first stage of labor when the vertex is meeting cervical resistance, or when the membranes rupture allowing flexion, then the next chance is when the presenting part reaches the inclined pelvic floor and the vertex is then in low pelvis, and the midpelvic plane and forceps are bypassed. Therefore, one need not expect

TABLE 3.—*Type of Forceps Used*

Type	Percentage
Simpson type (De Lee), Elliot, Ratzan	12.6
Solid blades (Tucker-McLane)	5.4
Williamson	3.6
Axis-traction (Bill's universal handle or Dewees)	54.8
Barton	2.6
Kielland	21.0
	100.0

spontaneous rotation in the midpelvis once the membranes have ruptured; it certainly is rare. If the vertex is in the occiput posterior or transverse position and if it will not descend below the midpelvis to the perineum, artificial rotation must be substituted in the great majority of cases. This failure of rotation is the indication for 40.8% of the midforceps operations. In seven out of every eight cases in which spontaneous rotation failed to occur, operative rotation was effected, whereas one out of every eight patients had to be delivered by traction to the direct occiput posterior position.

CHOICE OF FORCEPS

While the skill and experience of the operator are more important than the selection of the variety of forceps to be used, I do not subscribe to the view of some that a good forceps technician should be able to perform all varieties of forceps extraction equally well with the one instrument with which he is most familiar. The varieties of pelvic architecture and of pelvic contraction dictate that maneuvers should be so selected as to make use of the most favorable diameters.[4] If malposition exists, the decision to rotate at the level of arrest or above or below it is of great import.[5] In this study, six types of forceps were used.

4. Weinberg, A.: New York State J. Med. 40: 1530, 1940.
5. Weinberg, A.: West J. Surg. 48: 227, 1940.

PELVIC SIZE

There were no patients with contracted pelvic inlet who were delivered by midforceps. If a patient has a contracted inlet with a borderline or relative disproportion, and a trial of labor does not result in descent below the midpelvis, the patient is not delivered by midforceps but by abdominal section. In the recent study of midforceps deliveries by Steer,[6] about 70% were done for disproportion with a corrected fetal mortality of 3.8%.

There were 2.8% of contracted midpelvis in this series: those in which the interspinous was 9.5 cm. or less, or the posterior sagittal 4.0 cm. or less, none of which were associated with a contracted inlet. The association of a contracted inlet and midpelvis was considered to be an indication for abdominal delivery for a full term pregnancy.[7] In no case was a midforceps delivery of a full term pregnancy attempted when the midpelvic index (sum of the interspinous and posterior sagittal) was less than 13.5 cm.[8] There were six cases of contracted outlet (sum of bituberous diameter and posterior sagittal less than 16.5 cm.). The pelvic outlet index always equals or exceeds the midpelvic index so that it cannot be the definitive cause of disproportion unless there is a funnel effect caused by converging side walls, a narrow subpubic angle, and lower sacral variations. All cases of contracted pelvic outlet were associated with contracted midpelvis. When present, contraction of pelvic outlet and midpelvis contributed to arrest in low pelvis in those cases in which the vertex had passed the plane of least pelvic dimensions, that is, the midpelvis.

RESULTS

The results of any obstetrical procedure ultimately will decide its place in practice.

Mother.—Fortunately there was no maternal mortality. A total of 7.8% significant complications occurred, distributed as follows: sulcus tear 3.2; postpartum hemorrhage 1.6; urinary retention over two days 0.6; third degree perineal laceration 0.6; cervical laceration 0.6; postpartum infection 0.2; parametritis 0.2; Dührssen's cervical incisions 0.2; manual removal of placenta 0.2; phlebitis 0.1; inversion of uterus 0.1; paravaginal hematoma 0.1; fistula in ano 0.1.

Baby.—The fetal mortality was 0.6%. The six deaths were distributed during labor and after delivery as follows: intra partum death (during labor but before delivery), one; stillborn (death during delivery), two; neonatal deaths, three.

Intra partum death was caused by cord strangulation during descent due to three loops of cord around the neck.

Of the stillborn, the first was a 12 lb. baby (5,443.1 gm.) in which death was due to traumatic asphyxiation from impacted shoulders in a multipara. This fetal death was caused by disproportion due to an excessive sized baby in a very obese woman with a family history of diabetes, who is probably also a candidate for this disease. The second was in a primagravida with a 10 lb. (4,535.9 gm.) stillborn baby with a clinical birth injury caused by failure of forceps delivery and subsequently delivered by internal podalic version and breech extraction. An unrecognized malposition was the cause of the failure of forceps operation. Incidentally both of these patients had uncomplicated forceps deliveries with live babies at their next confinements; this rules out contracted pelvis as the cause of the stillbirths.

The three neonatal deaths were due to the following causes: (1) unrecognized occiput posterior with clinical birth injury, no autopsy being performed; (2) an intracranial hemorrhage proved by autopsy that occurred in an uncomplicated midforceps delivery; (3) unknown. Clinically there appeared to be no birth injury.

There were six nonfatal birth injuries all followed by complete recovery: cephalhematoma, two, or 0.2%; transient Erb's palsy, two, or 0.2%, and transient facial palsy, two, or 0.2%. As is well known, intracranial hemorrhage, Erb's palsy, and cephalhematoma may occur in spontaneous deliveries as well and in nearly the same proportion.

COMMENT

In this series of 1,000 midforceps deliveries in patients whose pelves were studied by pelviradiography either before or during labor, the maternal and fetal results were almost as good as could be expected in spontaneous deliveries, and much better than in any series of cesarean sections performed after labor began with which I am familiar.

The tendency in the current literature is to urge almost complete elimination of the midforceps operation. Steer's[6] results in his study of 227 midforceps operations were so poor that he recommended that it be seldom performed. The incidence of complications in his series was as follows: hemorrhage 15%, cervical lacerations 12%, Dührssen's incisions 3%, sulcus tear 23%, fistula 0.4%, maternal mortality 0.4%, and uncorrected fetal mortality 7% corrected to 5.3%.

The indications for the midforceps operation in Steer's series were divided into disproportion 70% and inertia 30%, whereas in my series the corresponding figures were 0.4% and 28%. By contrast, the fetal mortality in the disproportion group was 5.3% in his series, and 0.5% in mine. I never perform a midforceps operation in the presence of known disproportion. The deaths in this series were due to nonrecognition of excessive size babies in pelves of average size.

D'Esopo[9] states that only by reduction of the midforceps incidence to 0.5% and increase of the cesarean rate to 6% will it be possible to eliminate birth trauma to mother and babies and maintain a preventable term fetal loss of 1%. I have kept my preventable term fetal loss to 0.5% with a cesarean incidence of 3% and a midforceps rate of 11%. When one considers that the incidence of rupture of the uterus in subsequent pregnancies occurs in 1.7% and a very thin scar is found in another 2.3%, the later advantages of vaginal delivery are obvious. The immediate complications in 1,000 consecutive cases of cesarean section at Sloan Hospital for Women during the same period as my midforceps series were much higher: infection 10.7%, hemorrhage

6. Steer, C. M.: Am. J. Obst. & Gynec. **60**: 395, 1950.
7. Weinberg, A., and Scadron, S.: Am. J. Obst. & Gynec. **46**: 245, 1943.
8. Weinberg, A., and Scadron, S.: Am. J. Obst. & Gynec. **52**: 255, 1946.
9. D'Esopo, D. A.: Am. J. Obst. & Gynec. **59**: 77, 1950.

14.6%, wound infection 33%, wound disruption 0.2%, thromboembolism 1.8%, intestinal obstruction 0.2%, distention 23.6%, and respiratory 2.8%.

The major indication in my series for midforceps is malposition (40.8%). Rotation of a malposition in the absence of disproportion was performed 356 times without fetal mortality or serious injury. Fifty-two patients with malposition were delivered by midforceps without rotation. According to D'Esopo and Steer, these patients should have been subjected to further labor or section. At least 246 had the further trial of labor up to a full test. Prolonged second stage labor in itself is dangerous, and further trial might have compromised the baby and run the risk of uterine rupture. The other patients were delivered for miscellaneous indications; none required cesarean section as judged by the fetal and maternal results.

The relatively good results in this series are due, I believe, to the use of pelviradiography. Every patient had the benefit of this procedure either before or during labor. The roentgenographic pelvimetry ruled out pronounced pelvic contraction and almost eliminated cephalopelvic disproportion. The knowledge that, if a proper cephalic application is made, sufficient traction will result in delivery gives the operator a feeling of confidence. In only one case did the forceps fail to deliver, and that was because a cephalic application was not made owing to nonrecognition of the position. The knowledge of pelvic architecture aids in the proper selection and execution of the forceps operation. The roentgenogram, incidentally, in a number of cases was a check on a manual determination of station and position.

In cases of borderline midpelvic contraction with possible cephalopelvic disproportion at this plane, a full test of labor is given to allow the biparietal diameter to pass the ischial spines. This was determined by the presenting part being at or slightly below (1 cm.) the ischial tuberosities. If the vertex in an anterior position failed to reach this level, I suspected that the biparietal diameter could not pass through the plane of least dimensions, and a midforceps delivery was not attempted.

SUMMARY AND CONCLUSIONS

The midpelvis is defined as that portion of the true pelvis lying between the ischial spines and tuberosities. In a series of 1,000 midforceps operations in women who had the benefit of roentgenographic pelvimetry before or during labor, the uncorrected fetal mortality was 0.6%, corrected to 0.5%. There was no maternal mortality. The indication for the operation was arrest in 91.8%, of which 40.8% were due to malposition and 39.6% to poor contractions. Only 0.4% were due to disproportion. Distress accounted for 2.6%. The good results in midforceps operations performed in the absence of fetopelvic disproportion are indications for its retention rather than its almost complete replacement by cesarean section.

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PREVENTION OF RHEUMATIC FEVER BY PROMPT PENICILLIN THERAPY OF HEMOLYTIC STREPTOCOCCIC RESPIRATORY INFECTIONS

PROGRESS REPORT

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The role of the hemolytic Streptococcus in the precipitation of both initial and recurrent attacks of rheumatic fever is now well established. Furthermore, it is general experience that hemolytic streptococcic respiratory infections in children and young adults who have previously had one or more attacks of rheumatic fever cause recurrence of the disease in 40 to 50% of instances. When the usefulness of sulfonamides in the treatment of streptococcic infections was first discovered, attempts were made at the House of the Good Samaritan to prevent such recurrences by prompt treatment of hemolytic streptococcic infections in known rheumatic subjects with a sulfonamide preparation in the hope that, by suppression of the growth of streptococci in the upper respiratory tract, absorption into the blood stream of the streptococcic substance responsible for precipitating the rheumatic process would be reduced to a subeffective level. These attempts were not successful.[1] Subsequently, when penicillin became available, it seemed reasonable to believe that this antibiotic might be effective where sulfonamides had failed, the basis for this belief being that in vitro penicillin was found to be a more potent antistreptococcic agent than the sulfonamides and that in vivo penicillin was observed to be more effective than the sulfonamides in eradicating hemolytic streptococci from the throats of carriers and of persons with acute hemolytic streptococcic respiratory infections.[2]

From the House of the Good Samaritan (Children's Medical Center) Boston.

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Most of the oral penicillin used in this study was supplied by the Lederle Laboratories through Dr. Stanton M. Hardy.

1. Massell, B. F.: Studies on the Use of Prontylin in Rheumatic Fever, New England J. Med. **216**: 487 (March 18) 1937. Massell, B. F., and Jones, T. D.: The Effect of Sulfanilamide on Rheumatic Fever and Chorea, New England J. Med. **218**: 876-878 (May 26) 1938.

2. (*a*) Goerner, J. R.; Massell, B. F., and Jones, T. D.: Use of Penicillin in the Treatment of Carriers of Beta-Hemolytic Streptococci Among Patients with Rheumatic Fever, New England J. Med. **237**: 576-580 (Oct. 16) 1947. (*b*) Massell, B. F.; Dow, J. W., and Jones, T. D.: Orally Administered Penicillin in Patients with Rheumatic Fever, J. A. M. A. **138**: 1030-1035 (Dec. 4) 1948.

Consequently, in 1945 we began the studies that form the basis of the present report. Our preliminary observations were presented before the American Medical Association in June, 1948, as part of a broad study on various applications of penicillin to the rheumatic fever problem.[2b] Since that time, observations have been continued, and now we have accumulated data on a total of 46 proved clinical hemolytic streptococcic infections in subjects with proved rheumatic fever. Although not even this number of infections is sufficient to permit final conclusions, especially when consideration is given to the number of different serological species of streptococci involved in the infections, the possible practical application of our observations in relation to the problem of rheumatic fever prevention and the interest in such an application expressed at the recent National Conference on Cardiovascular Diseases[3] indicate the need of this progress report.

METHODS OF STUDY

The present report is based on observation on 46 clinical hemolytic streptococcic infections in 40 rheumatic subjects. Thirty-four persons had one infection each; whereas six persons had two infections at different times during the period of the study. With the exception of a 21-year-old girl the patients varied in age from 4 to 16 yr. The average age was slightly less than 10. Fifteen of the infections were in males; 31 were in females.

All the patients were proved rheumatic subjects, in that all gave a good history of one or more attacks of rheumatic fever. Furthermore, most of them had been observed by us personally at the time of at least one of the attacks, and 39 of the streptococcic infections occurred in persons with definite signs of rheumatic heart disease. Seventeen of the infections occurred in patients on the wards of the House of the Good Samaritan, seven in patients convalescing in one of the nursing homes of the Children's Mission to Children, and 22 occurred in ambulatory persons who were being observed carefully in connection with a so-called home study. The patients in the last group were examined in a clinic at weekly intervals and, in addition, were visited in their own homes at the time of all illnesses.

Base-line clinical and laboratory data had been collected on nearly all the patients for some time prior to the development of the streptococcic infections. The preinfection observations included throat cultures for hemolytic streptococci one to three times weekly and determination of the erythrocyte sedimentation rate and the antistreptolysin O titer of the serum at two to four week intervals. At the time of all febrile illnesses, whether or not these were associated with respiratory symptoms, the tests were repeated, and during the subsequent two months the frequency of the tests was increased. Throat culture swabs were planted and streaked directly on blood agar plates containing 5% horse blood. The antistreptolysin O titer was performed by a modification of the method of Hodge and Swift.[4]

In this study we have included only those infections with which there was fever as well as bacteriological evidence of invasion of the upper respiratory tract by hemolytic streptococci. In addition to fever, other symptoms and/or signs of pharyngitis or tonsillitis were usually, but not invariably, present. Pharyngeal or tonsillar exudate was not always observed.

The bacteriological evidence of streptococcic infection in most instances consisted in a change of the throat culture from negative to strongly positive for the hemolytic Streptococcus. In three patients there was a change in the throat culture findings from occasionally weakly positive prior to the clinical infection to strongly positive at the time of the infection. In several patients numerous hemolytic streptococci were cultured from the throat at the time of the clinical signs of infection, but preinfection cultures had not been taken. Subclinical hemolytic streptococcic respiratory infections, in which bacteriological evidence of infection was not accompanied by fever or other clinical manifestations of respiratory illness, were occasionally encountered, but, because of the difficulty in evaluation of these, they have not been included in the present study.[5]

Of the total of 46 clinical hemolytic streptococcic infections, 34 were treated with penicillin. For various reasons 12 infections were not treated with penicillin; these, therefore, served as controls.

In the treated group of patients, an attempt was made to begin therapy as soon as possible after the onset of clinical manifestations of infection; thus the administration of penicillin was usually begun immediately after the throat cultures had been taken and before the results of the cultures were known. Consequently, penicillin therapy was started in many suspected streptococcic infections in which the bacteriological findings failed to substantiate the diagnosis. In these instances, penicillin treatment was discontinued when the results of the throat cultures were obtained, and such infections were excluded from the study. In all streptococcic infections in which the diagnosis was confirmed by the bacteriological findings, penicillin treatment was continued for a total of 10 days. The time that elapsed between onset of symptoms and the beginning of penicillin therapy was usually less than 24 hr., occasionally 36 hr. and in one instance as long as 48 hr.

Five of the patients were treated intramuscularly with aqueous penicillin G in divided doses totaling 120,000 to 400,000 units per day. In the remaining patients, penicillin was administered by mouth in the form of buffered tablets of sodium or procaine penicillin G and usually in doses of 200,000 units five times daily (total of 1,000,000 units per day). For some patients treated at home it was found more practicable to change the dosage schedule after the first five days of treatment to 300,000 units three times daily. Care was always taken to prevent the mixing of penicillin with food by administration of the antibiotic at least one hour before meals and two to four hours after meals. Patients receiving penicillin were not allowed food between meals.

3. Proceedings of the National Conference on Cardiovascular Diseases, New York, American Heart Association, 1950.

4. Hodge, B. E., and Swift, H. F.: Varying Hemolytic and Constant Combining Capacity of Streptolysins: Influence on Testing for Antistreptolysins, J. Exper. Med. 58: 277-287 (Sept.) 1933.

5. Our preliminary report[2b] included five instances of penicillin-treated subclinical hemolytic streptococcic infections none of which was followed by rheumatic fever. Since the preparation of that report we have made no attempt to study subclinical infections; nevertheless, we are aware of two rheumatic fever recurrences which followed untreated infections of this kind.

An attempt was made to determine by the precipitin technique [6] the serological group and type of all strains of streptococci isolated at the time of the infections. Occasionally the strains were temporarily stored in the frozen state, and unfortunately in some of these instances the organisms did not remain viable and consequently could not be tested for their serological characteristics. Data regarding the frequency of group A strains among the hemolytic streptococci isolated from the 46 infections are presented in Table 1. Here it is shown that among the 34 penicillin-treated infections the causative organism was found to belong to group A in 25 instances. In four cases the isolated strains did not belong to group A, and in five instances the streptococci did not grow out and therefore could not be tested. Of the 12 infections not treated with penicillin, 11 were found to be due to group A streptococci.

TABLE 1.—*Data Regarding Frequency of Group A Strains Isolated from 46 Hemolytic Streptococcic Infections*

	Infections Not Treated with Penicillin	Infections Treated with Penicillin	Total Infections
Strains not isolated for grouping	1	5	6
Non-Group A strains	0	4	4
Group A strains	11	25	36
Total	12	34	46

Information regarding the serological type of the group A strains is given in Table 2. Of the 25 group A strains isolated from the penicillin-treated infections, 10 could not be typed with the available precipitin serums. These are referred to as type X. Of the 11 group A strains isolated from the infections not treated with penicillin, three were found to be type X.

TABLE 2.—*Serological Types of Group A Strains Isolated from 36 Streptococcic Infections*

Type	X*	2	3	4	5	6	12	22	30	41	Total Group A Strains
No. of infections not treated with penicillin	3	0	2	0	2	3	0	0	0	1	11
No. of infections treated with penicillin	10	3	1	1	6	1	1	1	1	0	25
Total infections	13	3	3	1	8	4	1	1	1	1	36

* Type X is used to designate those strains which could not be typed with the available serums.

FREQUENCY OF RHEUMATIC FEVER RECURRENCES

The frequency with which rheumatic fever recurrences followed the penicillin-treated and the untreated hemolytic streptococcic infections is indicated in Tables 3 and 4. In Table 3 the data have been analyzed without regard to the serological characteristics of the organism isolated at the time of the streptococcic infections, whereas in Table 4 the analysis has been restricted to infections demonstrated to be due to group A streptococci. It is evident that rheumatic fever recurrences followed approximately one-half of the untreated infections, an incidence which is consistent with our previous experience at the House of the Good Samaritan. In contrast, only two (6% to 8%) of the treated infections were followed by rheumatic fever.

In evaluation of these findings, attention should be called to the fact that in the penicillin-treated series of group A infections the isolated strains could not be typed with the available precipitin serums in 10 instances, and in two instances the streptococci were found to be either type 4 or type 22, two serological types that are believed by many investigators to be frequently nonrheumatogenic. In the untreated infections, type 4 and type 22 strains were not encountered; however, the organisms isolated from three of the infections could not be typed with the available serums. Both of the rheumatic fever recurrences in the penicillin-treated patients resulted from type 2 streptococcic infections. In the control series, recurrences followed streptococcic infections due to type 3 in one case, type 5 in two cases, type 6 in one case, and type X in two cases. None of the untreated infections was due to a type 2 Streptococcus.

Because of these variations in the serological characteristic of the organisms responsible for the respiratory infections and because of the total numbers of infections in the treated and especially in the control series, which are too small for a test of statistical significance, the results of this study should be interpreted with some reservations, and final conclusions should not be made until there has been an opportunity to observe a larger series of infections represented by a wider variety of serological types of streptococci.

TABLE 3.—*Frequency of Rheumatic Fever Recurrences Following Streptococcic Infections—Irrespective of Serological Group of Isolated Strains*

	No. of Infections	Recurrences	
		No.	%
Infections not treated with penicillin	12	6	50
Infections treated with penicillin	34	2	6

TABLE 4.—*Frequency of Rheumatic Fever Recurrences Following Streptococcic Infections—Proved Group A Infections Only*

	No. of Infections	Recurrences	
		No.	%
Infections not treated with penicillin	11	6	54
Infections treated with penicillin	25	2	8

Nevertheless, the difference in incidence of recurrences in the treated and untreated groups of patients is impressive and suggests that penicillin therapy of hemolytic streptococcic infections may be effective in preventing subsequent rheumatic fever. This impression is strengthened by the fact, previously mentioned, that prior to the use of penicillin at the House of the Good Samaritan hemolytic streptococcic infections were usually followed by rheumatic fever recurrences in 40% to 50% of instances. The clinical chart of one such rheumatic recurrence (Fig. 1) and, for comparison, the clinical chart of a patient successfully treated with penicillin at the time of a hemolytic streptococcic infection (Fig. 2) are shown.

A more detailed consideration of the clinical course and bacteriological findings in the two patients in whom rheumatic fever developed despite penicillin therapy supports the belief that penicillin played a role in the prevention of rheumatic fever in the remaining patients who

6. Swift, H. F.; Wilson, A. T., and Lancefield, R. C.: Typing Group A Hemolytic Streptococci by M Precipitin Reactions in Capillary Pipettes, J. Exper. Med. 78: 127-133 (Aug.) 1943.

were treated with this antibiotic. Both patients in whom therapy failed were convalescing in the same nursing home, and both experienced type 2 streptococcic respiratory infections within a short time of each other—one first became ill on Feb. 2 and the other on Feb 16, 1950. A type 2 clinical streptococcic infection also developed shortly afterward in a third patient who was

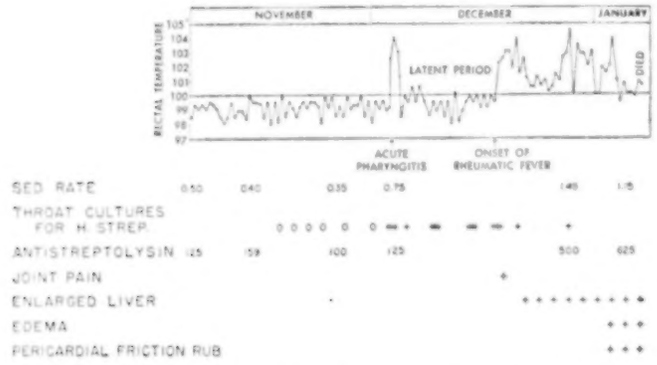

Fig. 1.—Clinical chart of a patient in whom a fulminating and fatal recurrence of rheumatic fever followed a hemolytic streptococcic infection. This episode was observed at the House of the Good Samaritan prior to the discovery of penicillin. Note presence of positive throat cultures and rise in antistreptolysin 0 titer.

treated with penicillin and in whom a rheumatic recurrence did not develop. Furthermore, during this same period, type 2 organisms were cultured from the throats of several other patients and personnel in the nursing home who did not present any clinical evidence of respiratory disease.[7]

Through a misunderstanding, the first patient in whom the type 2 infection developed was not treated with the usual oral dose of penicillin (200,000 units five times daily) until three days after the onset of his first symptoms. Instead, he received two doses of 200,000 units each on the evening of the first day (12 hours after the onset of clinical manifestations) and only 100,000 units five times during the second day. Despite this delay in the use of full penicillin dosage, the clinical respiratory manifestations subsided promptly and the throat cultures became negative for hemolytic streptococci. On Feb. 15, three days after discontinuation of penicillin therapy and 13 days after the onset of the initial streptococcic infection, the patient again experienced a high fever and the throat culture once more revealed the presence of numerous type 2 streptococci. This recurrent infection was treated within 12 hours with full oral doses of penicillin (200,000 units five times daily) and during a 10-day course of therapy the patient became clinically well and apparently free from streptococci. A throat culture taken five days after discontinuation of the second course of penicillin was reported as negative, but after another seven days (22 days after the second streptococcic infection) fever, joint pains, and other manifestations characteristic of rheumatic fever suddenly developed. A throat culture taken at this time revealed once more the presence of numerous type 2 streptococci. A clinical type 2 streptococcic infection developed in the second patient on the evening of Feb. 16, the day after the recurrence of infection in the previously described patient. This second patient received 200,000 units of penicillin orally within several hours after his first clinical symptoms and then, beginning the next morning, 1,000,000 units daily for another 10 days. Although there were no further clinical signs of respiratory infection, type 2 organisms reappeared in large numbers in the throat culture beginning one week after administration of the last dose of penicillin and were frequently cultured from the throat thereafter. About four weeks after the initial streptococcic infection and 10 days after the first reappearance of type 2 streptococci in the throat, the patient became ill with definite manifestations of rheumatic fever.

From the available facts it is not possible to determine whether the return of type 2 streptococci in the throats of these two patients after the discontinuation of penicillin therapy was due to reinfection contracted from other persons in the same house who harbored the organisms and to whom they were exposed or whether it was due to suppression but incomplete eradication of the streptococci from the respiratory tract during therapy and subsequent regrowth of the organisms, when they were no longer subject to the influence of the antibiotic. In either case it is evident that recurrent streptococcic infections developed in both patients, and it seems possible that these infections were responsible for the rheumatic fever which developed shortly thereafter.

Support for this interpretation is offered by the follow-up throat culture data obtained in 30 of the 32 remaining penicillin-treated patients, in none of whom did rheumatic fever develop. In 25 of these, the throat cultures became negative for hemolytic streptococci soon after the beginning of penicillin therapy and remained negative after the discontinuation of the antibiotic. In four cases, the throat cultures remained negative during therapy but occasional weakly positive cultures, indicating incomplete suppression of the organisms, were obtained after completion of treatment. In only one instance, in which the infecting organism was a somewhat penicillin-resistant nongroup A Streptococcus, did strongly positive cultures persist despite penicillin treatment. The bacteriological findings, then, in the "successful" penicillin-treated patients contrast strikingly with those in the two "failures" and suggest that the development of rheumatic fever in the latter patients was due to incomplete suppression of the organisms or to reinfection.

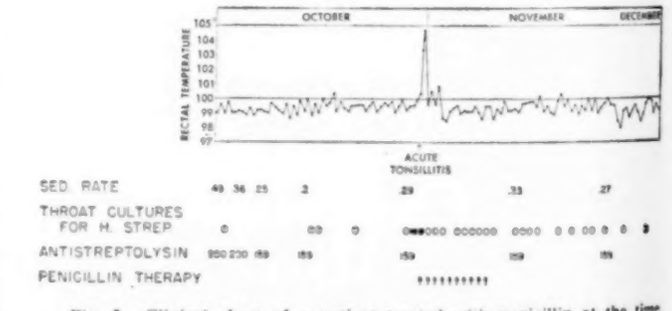

Fig. 2.—Clinical chart of a patient treated with penicillin at the time of a hemolytic streptococcic infection. This infection was not followed by an increase in rheumatic fever activity. Note prompt disappearance of streptococci from the throat flora and failure of antistreptolysin 0 titer to rise.

This interpretation, in turn, raises a question, which may be of practical importance but which cannot be

7. Drs. Rebecca C. Lancefield and Stuart D. Elliott of the Hospital of the Rockefeller Institute assisted in the identification of the type 2 strains.

answered without further study: Could the two instances of rheumatic fever recurrence have been prevented by more effective penicillin treatment (larger oral doses or intramuscular therapy), by treatment for longer than 10 days, or by the administration of penicillin to the other members of the house so that reexposure and reinfection might be prevented?

THE ANTISTREPTOLYSIN O RESPONSE

One of the toxins produced by hemolytic streptococci is an oxygen labile hemolysin known as streptolysin O. This substance is antigenic and, when released during streptococcic infection, stimulates the production of an antibody called antistreptolysin O. In the course of the present study a sufficient number of blood samples were obtained from 10 of the patients with untreated infections and from 27 of those with penicillin-treated infections, to allow for an accurate determination of the response of the antistreptolysin O titer.

Table 5, in which these observations are recorded, shows that a significant rise in titer (two tubes or more) followed all 10 (100%) of the untreated infections but only 10 (37%) of the penicillin-treated infections. A rise of four tubes or more was observed in nine (90%) of the untreated infections and in only four (15%) of those in which penicillin was administered. These data suggest that penicillin therapy of hemolytic streptococcic infections suppresses the antistreptolysin O response. Similar observations have also been made by other investigators.[8]

TABLE 5.—*Antistreptolysin O Response in 27 Infections Treated with Penicillin and in 10 Infections Not Treated with Penicillin*

Maximum Increase in Antistreptolysin O Titer (Measured in Tubes *)	0	1	2	3	4	5	6	7	8	9	10	Total Infections
Untreated infections	0	0	1	0	2	4	1	0	0	1	1	10
Penicillin-treated infections	14	3	4	2	2	1	1	0	0	0	0	27

* The antistreptolysin O test is so set up that the difference in the logarithms of the absolute titers in any two adjacent tubes is equal to 0.10.

The antistreptolysin O response, or, for that matter, the development of any other antibody, has not yet been definitely implicated in the mechanism by which rheumatic fever is precipitated by hemolytic streptococcic infections. Nevertheless, the fact that penicillin therapy can interfere with the elaboration of an antigen sufficiently to influence the antibody response is perhaps significant and suggests that this therapy similarly may interfere with the elaboration of the unknown streptococcic substance responsible for the production of rheumatic fever.

SIGNIFICANCE OF FINDINGS IN RELATION TO GENERAL PROBLEM OF RHEUMATIC FEVER PREVENTION

The problem of rheumatic fever prevention can be divided into two phases. The first is concerned with the prevention of recurrences; the second involves the prevention of initial attacks of the disease.

It is well known that rheumatic fever is a repetitive disease [9] and that with each new episode the degree of cardiac damage may be increased. Furthermore, the mortality from first attacks of rheumatic fever is relatively low, most deaths from rheumatic heart disease, at least in children and young adults, being traceable directly to recurrences of active rheumatic fever. Hence, anything that can be done to reduce the incidence of such recurrences should contribute toward the lowering of the mortality from and the reduction of the crippling effects of this disease.

The data that have been presented suggest that prompt and adequate penicillin therapy of hemolytic streptococcic respiratory infections in rheumatic subjects may be effective for this purpose. At the same time attention should be directed to the fact that approximately 40% to 50% of rheumatic episodes result from hemolytic streptococcic infections with which there are associated no fever or other clinical manifestations or in which symptoms are so mild that they are overlooked.[10] Obviously such infections, not being clinically recognizable, could ordinarily not be treated with penicillin and, therefore, even if prompt penicillin therapy of clinical hemolytic streptococcic infections should prove to be effective for rheumatic fever prevention, the maximum reduction in risk that could be expected by the method would be 50% to 60%.

Therefore, for childen and young adults who have had one or more previous attacks of rheumatic fever an attempt to prevent, in the first place, the development of all hemolytic streptococcic infections, subclinical as well as clinical, would seem to be a method of prophylaxis preferable to one which depends on the treatment of already developed infections. That the continuous daily administration of sulfadiazine or other sulfonamide in small doses may be useful for the prevention of streptococcic infections, and hence for the prevention of rheumatic fever recurrences, has already been demonstrated,[11] and data are now accumulating which suggest that daily oral penicillin may be even more effective for this same purpose.[12]

The second phase of the problem of rheumatic fever prevention is concerned with initial attacks of the dis-

8. Rantz, L. A.; Boisvert, P. J., and Spink, W. W.: Hemolytic Streptococcal Sore Throat: Antibody Response Following Treatment with Penicillin, Sulfadiazine (Sulfonamide) and Salicylates, Science **103**: 352-353 (March 22) 1946. Weinstein, L., and Tsao, C. C. L.: Effect of Types of Treatment on Development of Antistreptolysin in Patients with Scarlet Fever, Proc. Soc. Exper. Biol. & Med. **63**: 449-450 (Nov.) 1946. Kilbourne, E. D., and Loge, J. P.: The Comparative Effects of Continuous and Intermittent Penicillin Therapy on the Formation of Antistreptolysin in Hemolytic Streptococcal Pharyngitis, J. Clin. Investigation **27**: 418-424 (July) 1948. Weinstein, L.; Bachrach, L., and Perrin, T. S.: Studies of the Influence of Penicillin on the Immune Reactions in Streptococcal Pharyngitis, J. Clin. Investigation **28**: 817-818 (July) 1949.

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11. Thomas, C. B., and France, R.: A Preliminary Report of the Prophylactic Use of Sulfanilamide in Patients Susceptible to Rheumatic Fever, Bull. Johns Hopkins Hosp. **64**: 67-77 (Jan.) 1939. Coburn, A. F., and Moore, L. V.: The Prophylactic Use of Sulfanilamide in Streptococcal Respiratory Infections, with Especial Reference to Rheumatic Fever, J. Clin. Investigation **18**: 147-155 (Jan.) 1939. Kuttner, A. G., and Reyersbach, G.: The Prevention of Streptococcal Upper Respiratory Infections and Rheumatic Recurrences in Rheumatic Children by the Prophylactic Use of Sulfanilamide, J. Clin. Investigation **22**: 77-85 (Jan.) 1943. Rosenberg, E. F., and Hench, P. S.: Recent Advances in the Treatment of Rheumatic Fever, with Special Reference to Sulfonamide Prophylaxis and Intravenous Salicylate Therapy, M. Clin. North America **30**: 489-509 (May) 1946.

12. (*a*) Massell, Dow and Jones.[2b] (*b*) Massell, B. F.: Effect of Orally Administered Penicillin on the Carriage of Hemolytic Streptococci: Significance in Relation to Use of Penicillin for Rheumatic Fever Prophylaxis, to be published.

ease. If penicillin treatment of hemolytic streptococcic infections suppresses the growth of streptococci in the upper respiratory tract sufficiently to reduce the elaboration of the unknown rheumatic fever-producing substance to subeffective levels in persons who have previously had one or more attacks of rheumatic fever and who are presumably unusually vulnerable to the disease, it seems reasonable to expect that similar treatment would have a similar effect in persons who are susceptible to rheumatic fever but who have not yet had an attack. The validity of this inference is supported by the recent report of Denny and co-workers,[13] who observed in an air force training base only two cases of definite rheumatic fever in 798 young men with streptococcic infections treated with penicillin in contrast to 17 definite attacks of the disease in 804 untreated patients. On the other hand, Weinstein and his associates [14] observed rheumatic fever in 12 (7%) of 167 scarlet fever patients treated with penicillin and concluded that "the treatment of scarlet fever with crystalline penicillin G does not prevent the development of rheumatic fever."

Although these latter studies raise some doubt about the value of penicillin therapy of streptococcic infections for rheumatic fever prevention, attention should be called to the fact that most of Weinstein's cases of rheumatic fever were extremely mild and in many the diagnosis rested on slight changes in serial electrocardiograms. The extreme mildness of the disease process in these patients is indicated especially by the fact that in all but one the sedimentation rate was not elevated, a finding which contrasts strikingly with our experience at the House of the Good Samaritan, where, except in the presence of congestive heart failure, normal sedimentation rates in patients with other clear evidence of active rheumatic fever have been observed rarely. Even if Weinstein's criteria for rheumatic fever are accepted, the possibility must be considered that the incidence of a similar mild rheumatic process would have been a great deal higher than 7% in his scarlet fever patients had they not been treated with penicillin.

From the foregoing discussion it is evident that further observations will be necessary before any final conclusions can be drawn regarding the value of treating hemolytic streptococcic infections as a method for preventing initial attacks of rheumatic fever. Nevertheless, the available data are sufficiently suggestive to warrant optimism about the results which will be revealed by further studies.

SUMMARY AND CONCLUSIONS

Rheumatic fever recurrences in rheumatic subjects developed after only two out of a total of 34 hemolytic streptococcic infections in which penicillin therapy was begun promptly and continued for 10 days. In contrast, there were six instances of rheumatic fever following 12 similar infections in which penicillin therapy was not used. The administration of penicillin also appeared to suppress the antistreptolysin 0 response in many cases. These observations suggest that prompt and adequate penicillin therapy of hemolytic streptococcic infections in rheumatic subjects is effective in reducing the incidence of rheumatic recurrences. However, because of the relatively small number of cases involved in this study and because of the variety of serological groups and types of streptococci associated with the infections, final conclusions are not yet warranted. The observations also support other investigators' studies which suggest that the incidence of initial attacks of rheumatic fever can be reduced by penicillin therapy of clinical streptococcic respiratory infections. Prompt penicillin therapy of streptococcic infections is not recommended as a substitute for the use of continuous prophylactic measures in persons who have had one or more attacks of rheumatic fever.

25 Binney St.

13. Denny, F. W.; Wannamaker, L. W.; Brink, W. R.; Rammelkamp, C. H., Jr., and Custer, E. A.: Prevention of Rheumatic Fever: Treatment of the Preceding Streptococcic Infection, J. A. M. A. **143**: 151-153 (May 13) 1950.

14. Weinstein, L.; Bachrach, L., and Boyer, N. H.: Observations on the Development of Rheumatic Fever and Glomerulonephritis in Cases of Scarlet Fever Treated with Penicillin, New England J. Med. **242**: 1002-1010 (June 29) 1950.

RHEUMATIC HEART DISEASE IN PATIENTS OVER SIXTY YEARS OF AGE

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and
Charles E. Kossmann, M.D., New York

Rheumatic fever and rheumatic heart disease are usually considered to be diseases of the young. The average age of onset of rheumatic fever has been calculated in several series of cases with rheumatic heart disease. In 3,129 deceased patients studied by Cohn and Lingg [1] it was 14.5 ± 0.3 years for males and 15.0 ± 0.3 years for females; in 644 patients observed by De Graff and Lingg [2] it was 17 years; in 1,042 children observed by Wilson and Lubschez [3] it was 6.5 years. There are reports, however, of initial episodes of rheumatic fever in the seventh decade.[4] Among 3,741 patients with rheumatic fever entering Bellevue Hospital from 1911 to 1919, Lambert [5] found 91 over the age of 60.

Older data on the duration of life after the initial manifestation of rheumatic fever in the course of rheumatic heart disease indicate a poor prognosis. Cohn and Lingg [1] found the mean duration of life to be 13 years; De Graff and Lingg,[2] 15 years. In contrast to these, Wilson and Lubschez's study [3] demonstrated that the median duration of life for rheumatic children (with cardiac involvement) is greater than 30 years after onset. The discrepancy between the first two reports and the last is

This article has been shortened for publication in THE JOURNAL by omission of tables 1, 2, and 3. These will appear in the authors' reprints.

From the Department of Medicine, New York University College of Medicine; the Adult Cardiac Clinic and wards of the Third (New York University) Medical Division, and the Pathological Laboratories of Bellevue Hospital.

attributable to the selection of different groups of rheumatic patients. As Swift and Cohn [6] noted:

> . . . De Graff's cases, from Bellevue Hospital, were originally chosen because they had reached the stage in their cardiac career where they had symptoms of cardiac insufficiency from which they subsequently died; rheumatic children who died early as well as those who later did not develop cardiac symptoms were not included.

The latter constitute possibly 30 to 50 per cent of young rheumatic cardiac patients.[7] They would also be excluded from studies such as reported by Wilson and Lubschez and by Cohn and Lingg. Heart disease in them is usually discovered by routine thoracic x-rays and by physical examinations made for some other purpose. In some the discovery of gross rheumatic lesions is an incidental finding at necropsy.[8] Microscopic evidence of rheumatic cardiac lesions is even more often an incidental finding, the frequency depending somewhat on the pathological criteria for rheumatic disease used.[9]

Although the average duration from onset to termination of rheumatic heart disease is relatively short, there are extremes on either side of the mean. Some patients die in heart failure during their first bout of rheumatic carditis or within a decade of its onset. Others recover, have no recurrences, and survive for 40 years or more with valvular disease; they ultimately die of an unrelated disease without ever having had myocardial insufficiency.[10] Isolated cases with an interval varying from 54 to 63 years between the initial attack of rheumatic fever and the final clinical observations have been reported.[11] In one series of 12,000 patients [1] there were 75 who died at 60 years of age or over, and 25 patients over 60 years of age were still living at the time of the report.[12]

White and Bland [13] explained the benign clinical course of rheumatic heart disease with long survival on the basis of (1) a relatively mild degree of mitral stenosis; (2) the development of rheumatic heart disease in later life; (3) the relatively quiet, careful mode of living, and (4) probable ancestral longevity. Other reasons for longevity often given are the development of hypertension (Baker and Musgrave,[4] and Cookson [14]); the infrequency or mildness of recurrent rheumatic activity (Cohn and Lingg,[1] Baker and Musgrave,[4] and Wilson [15]); the absence of atrial fibrillation (Cookson [14] and Kossmann and Connor [16]); involvement of the mitral valve to the exclusion of other valves (Cabot [17]); the presence of chorea as the sole rheumatic manifestation (Cohn and Lingg [1]).

The present study was undertaken for three reasons: (1) to find, if possible, the causes for longevity in a selected group of patients with rheumatic heart disease and over 60 years of age; (2) to learn what clinical data, if any, might be of use in recognizing rheumatic heart disease so often overlooked in the aged; (3) to dispel the common clinical notion that advanced age and rheumatic heart disease are incompatible.

METHODS

The 71 patients in the study were observed in the Adult Cardiac Clinic and on the wards of the Third Medical Division of Bellevue Hospital. A few were private patients seen in consultation. Criteria used in their selection were (1) the presence of clinical or necropsy evidence of gross rheumatic heart disease; (2) age of 60 years or over at the time of the last observation or at the time of death.

The criterion used in the clinical diagnosis of rheumatic heart disease was the finding of physical and laboratory evidence of a characteristic structural lesion in the heart.[18] In all but four this "characteristic" structural lesion was mitral stenosis with or without other valvular disease. In the four exceptions there was clinical evidence of aortic valvular disease alone (cases 7, 12, 19, and 28), a good history of rheumatic fever, and discovery of heart disease in three early in life. In 48 patients there was a history of rheumatic fever; in all but 6 one of the major manifestations, namely polyarthritis, chorea, or carditis, had occurred. The pathological criteria for rheumatic disease, present in all of the 20 patients who came to necropsy, were those defined by de la Chapelle, Graef, and Rottino.[19] Actually, 14 of these were included in the report made by these authors.

RESULTS

Clinical and Pathological Results.—The case summaries have been arranged in three groups, each in the order of decreasing age of the patients. The first includes 36 patients alive at last observation (table 1); the second includes 15 patients who died but did not come to necropsy (table 2); and the third includes 20 patients who came to necropsy (table 3). Table 4 is an over-all summary of the three groups.

Six patients in the series had rheumatic heart disease for 60 years or longer. Four of these were alive and well at last observation. One patient (M.Do.), 80 years of age, had chorea and "irregular heart action" at the age of 14, and polyarthritis at 22 and 31 years. She had nine normal pregnancies and one miscarriage, and is still physically active.

In 48 patients (68%) in whom there was a history of a rheumatic manifestation in the past (carditis, polyarthritis, chorea, scarlet fever, or "rheumatic fever"), the average age of onset was 21.5 years; the average age at time of death or at the time of the last clinical observation was 66.9 years, and the mean duration from the onset of rheumatic fever to the last recorded age was 43.6 years. The average age of onset was therefore 4.5 to 15

1. Cohn, A. E., and Lingg, C.: The Natural History of Rheumatic Heart Disease: A Statistical Study; Onset and Duration of Disease, J. A. M. A. **121**: 1 (Jan. 2) 1943.

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years later than in previously reported series (Cohn and Lingg,[1] De Graff and Lingg,[2] and Wilson and Lubschez[3]), but the scatter was considerable (from 5 years to 55 years of age). Statistical proof of the significance of the difference in means of age at onset of the present series and of the series of Cohn and Lingg's deceased patients was obtained by the method of unpaired comparisons using the "standard deviation of each sample" method. The ratio (t) of the difference between the means to the standard error of this difference was 3.84 in the case of the males of Cohn and Lingg's series, and 3.57 in the case of the females. Both values are greater than the probability level of 2.6 of the normal curve ($P<1\%$), and therefore are highly significant statistically. This means that the later mean onset of rheumatic fever in the present series was very probably a determining factor in longevity.

Ancestral longevity as a factor in determining long life with rheumatic heart disease is most difficult to evaluate. The average age of the mother was 60.7 years in 29 instances; the average age of the father was 64.9 years in 29 instances. Of the total of 58 parents, two-thirds were over 60 years of age. With few exceptions, these averages represented the age of death of the parents. There were six patients with one parent and two patients with both parents who were over 80 years of age. A conclusion from these data cannot be drawn without recourse to a lengthy statistical analysis which should include several types of control groups.

Recurrences of rheumatic fever were not inconsiderable (tables 1, 2, and 3). There were 16 patients (30% of those with a history of a rheumatic manifestation) who had multiple attacks of rheumatic fever in some form. Four had only two attacks. One had the maximum number of six; this patient (J.Ho.) was last examined at the age of 63, 31 years after the first attack (table 1). The high frequency of recurrences in the series suggests that recurrences alone were not related to longevity, although this is not absolute. Perhaps the relatively older average age of recurrences, just as the older average age of onset, was the important factor. There was, of course, no way of evaluating the significance of severity of initial or subsequent attacks of rheumatic fever.

Only 2 patients had chorea, usually regarded as a mild manifestation so far as the heart is concerned.[1] However, both of these had, in addition, polyarthritis during other attacks.

The varieties of valvular lesions were of interest particularly since it is believed by some (Levine,[10] Rothschild, Kugel and Gross,[19] Criteria Committee of the New York Heart Association [18] and Stewart and associates [20]) that certain combinations alter the hemodynamics in a favorable way. Of the 71 patients (table 6), 42 (59.1%) had mitral stenosis and insufficiency; 22 (30.9%) had combined mitral and aortic lesions; three had combined mitral and tricuspid lesions; and four had aortic disease alone. In the 20 necropsied cases none displayed aortic lesions alone (table 5). It is difficult to conclude that any one lesion or combination of lesions accounted for longevity since similar distributions occur in any age group. To be noted particularly is the paucity of patients with tricuspid stenosis (2 proved, 1 questionable), and the presence of a tight mitral valve in several (table 3, cases 4, 6, 9, 14, and 17). Although table 5 shows a smaller mean weight of the hearts with tricuspid stenosis and a larger mean weight with aortic valve disease as compared to the weight of hearts displaying mitral lesions alone, it is probable that the total number of cases of each type is too small to draw conclusions.

Atrial fibrillation is also regarded as a terminal event in the disease, although durations in excess of 20 years have been reported (Kossmann and Connor [16] and Bishop and Bishop, Jr.[21]). In the present series, 33 patients displayed this arrhythmia at some time. Of these the duration was fairly certain in 16, where it averaged five years. However, 9 patients displayed the rhythm for more than five years, and the maximum duration was 14 years. Other abnormalities encountered electrocardiographically are shown in table 6.

TABLE 4.—*Summary of Course of Rheumatic Heart Disease in Seventy-One Patients Over Sixty Years of Age*

(1)	(2)		(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
Group and Number	Sex		Average Age	Age of Onset of Rheumatic Fever	Age of Diagnosis of Rheumatic Heart Disease	Age of First Diminution in Cardiac Reserve	Age of First Bout of Congestive Heart Failure	Age Digitalis Begun	Duration from Onset (3)-(4)	Duration from First Diminution in Cardiac Reserve (3)-(6)	Duration from First Bout of Congestive Heart Failure (3)-(7)	Duration from Onset Rheumatic Fever to First Diminution in Cardiac Reserve (6)-(4)
	Male	Female										
1 (36 living)	14	22	68.2	17.3	44.4	50.2	57.9	56.2	47.4	15.3	9.6	37.7
2 (15 dead—no necropsy)	10	5	64.3	28.4	54.4	56.4	58.5	62.5	36.6	9.2	7.2	29.3
3 (20 dead—necropsy)	13	7	66.4	25.0	60.7	60.4	62.1	62.9	41.6	7.7	4.7	35.3
Mean	..	..	66.9	21.5	50.7	64.4	59.5	59.7	43.6	12.2	7.9	30.6
No. cases in mean	..	..	71	48	64	62	51	48	48	51	31	47

10. Levine, S. A.: The Great Importance of Valvular Injury in Rheumatic Heart Disease, in Libro Homenaje al Profesor Doctor Ignacio Chavez, Mexico, D. F.: La Universidad Nacional Autonoma Mexico, 1945, p. 185. Peacock, T. B.: On Some of the Causes and Effects of Valvular Diseases of the Heart, in the Croonian Lectures of the Royal College of Physicians, 1865, London, J. & A. Churchill, Ltd., 1865, p. 108. Rothschild, M. A.; Kugel, M. A., and Gross, L.: The Incidence of Active Infection in Cases of Rheumatic Cardiovascular Disease During the Various Age Periods, Am. Heart J. **9**: 586, 1934.

11. White, P. D.: Optimism in the Treatment of Cardiovascular Disease, in Contributions to the Medical Sciences in Honor of Dr. Emanuel Libman by His Pupils, Friends and Colleagues, New York, International Press, 1932, vol. 3, p. 1205. Willius, F. A., and Smith, H. L.: Factors Concerned in Cardiac Hypertrophy: A Study Made at Necropsy of 79 Cases of Rheumatic Heart Disease, Am. Heart J. **10**: 190, 1934.

12. Lingg, C.: Personal communication to the authors.

13. White, P. D., and Bland, E. F.: Mitral Stenosis After 80, with Especial Reference to Dr. Herman F. Vickery, J. A. M. A. **116**: 2001 (May 3) 1941.

14. Cookson, H.: Mitral Stenosis in Later Life, Brit. Heart J. **11**: 155, 1949.

15. Wilson, M. G.: Rheumatic Fever, New York, Commonwealth Fund, Division of Publications, 1940, p. 249.

16. Kossmann, C. E., and Connor, C. A. R.: Auricular Fibrillation of Long Duration in Rheumatic Heart Disease, Am. Heart J. **24**: 215, 1942.

Only 9 patients (table 7) had a blood pressure persistently above 150 mm. Hg. systolic and 90 mm. Hg. diastolic. Six of these displayed atrial fibrillation. Hypertension, therefore, was not a frequent finding (7.9%) in the series.

A point of interest from the viewpoint of differential diagnosis was the mean duration of diminished cardiac reserve (table 4) which in 51 patients was 12.2 years (maximum 53, minimum 0). The mean duration of life after the first bout of heart failure was 7.9 years (maximum 30, minimum 0). It was the impression that these relatively large values would be helpful in suspecting the disease clinically. To this end a comparison was made with similar values determined in a group of 138 patients with degenerative (hypertensive and/or arteriosclerotic) heart disease. These lived on the average 1.8 years after the appearance of the first evidence of congestive heart failure, although 24% were alive three years after.[22] These figures would seem to substantiate the clinical impression that rheumatic heart disease is to be suspected when the course is characterized by prolonged cardiac disability.

TABLE 5.—*Summary of Valvular Lesions and Heart Weight in Eighteen Necropsied Cases* * †*

Diagnosis (Anatomical and Clinical)	MS, MI	MS, MI, AS, AI	MS, MI, TS
Male	8	5	—
Female	5	—	2
Total	13	5	2
Average heart weight in grams	527	677	450

* Two cases are not included because the weight of the heart was omitted in the autopsy protocol.

† Abbreviations used in this table: AI, aortic insufficiency; AS, aortic stenosis; MI, mitral insufficiency; MS, mitral stenosis; TS, tricuspid stenosis.

TABLE 6.—*Summary of Electrocardiographic Abnormalities in Relation to Valvular Lesions* *

	Total	MS, MI	MS, MI, TS	MS, MI, AI	MS, MI, AS, AI	AS, AI	AI
Total patients	71	42	3	13†	9	2	2
Male	38	18	..	9	8	1	2
Female	33	24	3	4	1	1	..
Normal sinus rhythm	20	11	..	4	3	2	..
Auricular fibrillation	33	21	3	7	2	..	..
Auricular flutter	1	1	..	..	..	..	..
Auricular tachycardia	1	..	..	1	..	..	..
Paroxysmal auricular fibrillation	1	..	..	..	..	1	..
Sinus tachycardia	1	1	..	..	..	..	..
Sinus bradycardia	3	1	..	..	..	..	2
Bundle branch block—right	2	2	..	..	..	..	..
Bundle branch block—left	1	1	..	..	..	..	..
Bundle branch block—type unclassified	1	..	..	..	1	..	..
Deviation of electrical axis—none	30	21	1	3	3	..	..
Deviation of electrical axis—right	5	3	1‡	1	..	..	..
Deviation of electrical axis—left	23	10	1	7	1	2	2

* Abbreviations used in this table are explained in table 5.

† Patient D. Co. (living, no. 4), questionable aortic stenosis.

‡ Patient L. Re. (living, no. 30), questionable tricuspid stenosis.

It was not possible to determine the causes of heart failure in the group. In this regard the hearts of only three patients displayed evidence of rheumatic activity at necropsy.[9] This was out of a total of 12 which were examined microscopically. This is comparable to a recently reported series of elderly rheumatic cardiacs [23] in which four of 16 patients over 60 years old showed lesions of rheumatic activity at necropsy.

A point of considerable clinical interest is that the diagnosis of rheumatic heart disease, particularly of mitral stenosis, was missed during life in eight of the 20 patients who came to necropsy (table 3).

Electrocardiographic Results.—The standard leads were available in 58 patients, precordial leads in a few, and intracardiac leads in one. The findings are summarized in table 6. Of greatest interest is that only five of the 58 displayed right deviation of the axis of QRS (angle alpha greater than + 90 degrees or less than — 90 degrees), and only two displayed right bundle branch block (patients M.Ca. and S.Pa.). One patient (F.Mc.) who displayed a right ventricular wall 1.2 cm. thick at necropsy, showed a right deviation of the mean axis of QRS, and a low voltage rsR′ in the lead from the right sternal edge but the QRS interval was 0.08 sec. An insufficient number of precordial leads were available in other cases to make any further statements about them. It is often stated that disease of the mitral valve among other things should be suspected in older patients if the electrocardiogram displays right deviation of the electrical axis of QRS. This is probably true but the present electrocardiographic observations indicate that the diagnosis will be missed in the majority of older patients with rheumatic mitral stenosis if too much reliance is placed on this finding.

TABLE 7.—*Analysis of Nine Cases with Hypertension*

	MS, MI*	MS, MI, TS*	MS, MI, AI*	MS, MI, AS, AI*	Total
	3	1	3	2	9
Male	1	..	2	2	5
Female	2	1	1	..	4
Age onset rheumatic fever	10 (unknown in 2)	Unknown	14 (unknown in 2)	18 (unknown in 1)	10, 14, 18 (unknown in 6)
Left axis deviation	..	..	1	1	2
Right axis deviation	..	1	..	..	1
No deviation electrical axis	3	..	1	..	4
Auricular fibrillation	3	1	2†	1	7
Normal sinus rhythm	..	..	1	1	2

* Abbreviations used in this table are explained in table 5.

† 1 paroxysmal.

SUMMARY AND CONCLUSIONS

The clinical course of 71 patients over 60 years of age with rheumatic heart disease was reviewed for the purposes of determining the factors responsible for their

17. Cabot, R. C.: Facts on the Heart, Philadelphia and London, W. B. Saunders Company, 1926, p. 143.

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19. de la Chapelle, C. E.; Graef, I., and Rottino, A.: Studies in Rheumatic Heart Disease, Am. Heart J. **10**: 62, 1934.

20. Stewart, H. J.; Deitrick, J. E.; Watson, R. F.; Wheeler, C. W., and Crane, N. F.: The Effect of Valvular Heart Disease on the Dynamics of the Circulation, Am. Heart J. **16**:477, 1938.

21. Bishop, L. F., and Bishop, L. F., Jr.: Auricular Fibrillation Observed for 25 Years, J. A. M. A. **114**: 955 (March 16) 1940.

22. Goldring, W., and Chasis, H.: Hypertension and Hypertensive Disease, New York, The Commonwealth Fund, Division of Publications, 1944, p. 26.

23. Kaufman, P., and Poliakoff, H.: Studies on the Aging Heart: I. The Pattern of Rheumatic Heart Disease in Old Age (A Clinical-Pathological Study), Ann. Int. Med. **32**: 889, 1950.

longevity and of noting any manifestations which might make the clinician suspect the presence of the disease.

Of those possible factors in longevity which could be analyzed, including the degree of mitral stenosis, ancestral longevity, accompanying hypertension, infrequent recurrences of rheumatic fever, absence of atrial fibrillation, univalvular involvement, and late age of onset of rheumatic fever, only the last seemed to be a statistically valid factor in determining long life with rheumatic heart disease.

The mean duration of diminished cardiac reserve in 51 patients was 12.2 years; the mean duration of life after the first bout of congestive heart failure was 7.9 years. Therefore, the disease should be suspected in elderly patients when cardiac disability persists over a period of years.

The high incidence (40%) of missed diagnoses in the group of 20 patients that came to necropsy suggests that the incidence of rheumatic heart disease in the aged is far greater than available data indicate.

FAILURE OF TONSILLECTOMY

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Tonsillectomy is the commonest surgical operation, constituting about one-fourth of all operations performed. It is the operation that is done by more different physicians than any other. It is not usually a successful operation, however, because residual tonsil tags were found in 68% of 1,000 tonsillectomized patients. Better results can be achieved by improvement of the technique of the operation.

THE STUDY

One thousand patients were observed consecutively without selection in a general surgical practice over a period of years. Every patient who had had a tonsillectomy was interrogated and examined. They were asked (1) their age, (2) the age at tonsillectomy, (3) where the operation was done, (4) whether the surgeon was a "nose and throat" specialist, (5) why the operation was performed, (6) what anesthetic was used, (7) whether any benefit was obtained, and (8) whether to their knowledge any tonsillar tissue was left. Then the pharnyx was carefully examined with the use of a good light, a metal tongue depressor, and a tonsil pillar retractor. The tonsil tags were recorded as grade one to four, grade one being the smallest and grade four being an amount comparable to a normal size tonsil. The presence of lymphoid tissue not identifiable as tonsil tissue was also recorded.

THE RESULTS

Sixty-three per cent of the 1,000 patients were women. The average age was 36; the average age at operation was 19. Sixty patients had had two operations, and three had had three tonsillectomies. Only 240 thought that their surgeon was a specialist. Geographically the operations were performed mostly in the Pacific Northwest and near Texas. Sixty per cent of the operations were done with the patients under general anesthesia. Nineteen per cent thought their tonsils had been removed to improve their general health, and the other 81% because of local throat disorders. Thirty-one per cent denied any beneficial effect from the operation. Forty-two per cent of those expecting to have their general health improved denied receiving any benefit. Among those denying benefit, the incidence of retained tonsil tags was no greater than among those claiming benefit from the operation. The indications for tonsillectomy as revealed by this group of patients were very broad. This part of the study, of course, is largely subjective and probably is merely interesting and not valid scientific evidence justifying any conclusion regarding tonsillectomy.

Objectively, 68% of the patients had tonsil remnants, and only 32% had had a complete tonsillectomy. Of the 2,000 tonsil fossae examined, 1,120, or 56%, had tonsil tags. Forty-nine per cent of these tags were grade one, 29% were grade two, 14% were grade three, and in 8% the remnants were as large as a normal tonsil (grade four). Thirty-three per cent of the 1,000 patients had lymphoid tissue not identifiable as tonsil tissue. Among the 32% with no tags, some lymphoid tissue was found in the tonsillectomy scar area in 26%. Only 20% of the patients had throats that were absolutely clear of all lymphoid tissue in the palatine tonsil area. Only 20% of the patients with tags were aware of their existence. One per cent of those with clean throats thought they still had some retained tonsil tissue. Among the 400 patients who had had a local anesthetic, only 59% had tags. This would appear to be a significant difference in favor of a local anesthetic but may merely reflect the comparative ease of operative procedure in the larger throats of an older age group. Among those who believed their surgeon was a specialist, 64% had retained tags, a percentage that is not impressively better than the general average.

THE LITERATURE

These results are consistent with other similar studies. Gooch and Lillie [1] of the Mayo Clinic have recently published an excellent study of tonsil tags. They found an incidence of 22% of tonsil tags among the patients who had had a tonsillectomy prior to registration at the clinic. Sputh and Sputh [2] reported an incidence of 80% of tags among 285 patients. Kaiser [3] found an incidence of 38% in 1,000 school children. He also observed the incidence of otitis media, sore throats, and other diseases commonly associated with chronically infected tonsils to be increased in those with incomplete operations. His classic work is the only study found of the effect of tonsillectomy that considered the factor of incomplete operation.

Read before the Annual Meeting of the Oregon State Medical Society, Gearheart, Ore., Sept. 27-29, 1950.

1. Gooch, J. O., and Lillie, H. I.: A Clinical and Pathologic Study of Tonsil Tags, Ann. Otol., Rhin. & Laryng. **57**: 957 (Dec.) 1948.

2. Sputh, C. B., Jr., and Sputh, C. B.: The Tonsil Stump and Its Removal, J. Indiana M. A. **39**: 401 (Aug.) 1946.

3. Kaiser, A. D.: Results of Tonsillectomy, J. A. M. A. **95**: 837 (Sept. 20) 1930.

Lüscher [4] noted an incidence of 22% in 224 patients he had operated on himself. Campbell [5] observed an incidence of 77% in 631 tonsillectomized university students. McLaggan [6] reported an incidence of 49% in 100 school children. He titled his article "A Plea for More Careful Tonsillectomy." Rhoads and Dick [7] found an incidence of 73% among a group of 403 nurses. Clark [8] in 1913 reported 43% among 143 patients operated on at the Massachusetts General Hospital. Coues [9] reported in 1910 an incidence of 30% in 40 school children.

The variation in the percentage of tags found by the various reporters is probably due to variation in the criteria for identification of a tonsil tag. Gooch and Lillie [1] reported that otolaryngologists found half again as many tags in a given group of clinic patients as had the general diagnosticians. McLaggan only considered fragments as large as one-fourth of the tonsil. Most reporters did not discuss their criteria. In this study the presence of a crypt in a mass of lymphoid tissue in the tonsillectomy scar area was taken as the identifying characteristic of tonsil tissue. The single characteristic that distinguishes the palatine tonsil from the other lymphoid tissue of the pharynx is the crypt. Lymphoid tissue of the adenoid or lingual regions does not contain crypts. Inflammation often facilitates identification by accentuation of the crypt. It is probable that some of the patients reported to have lymphoid tissue actually had tonsillar remnants. None of the other reporters distinguished between lymphoid tissue and tonsil remnants.

The patients themselves usually assumed that their tonsils had "grown back." I have never observed any such regrowth. Most of those who have studied the problem agree that tonsils do not recur.[10] Some [11] have remonstrated that all the extra capsular lymphoid tissue of the tonsil area cannot possibly always be removed. It is probably true that there is commonly considerable extra capsular lymphoid tissue. This peritonsillar lymphoid tissue probably is not tonsil tissue.[12] However, my own careful dissections have demonstrated the frequent existence of irregular rounded lobules of tonsil 2 to 20 mm. in diameter extending out beyond the so-called capsule into the peritonsillar tissue. These parts of the tonsil can easily be left in place by rough, careless dissection. Some tags probably represent such lobules left in place. Most of the tags are at the top or bottom of the tonsil and are plainly bits of the visible tonsil not disturbed by crude dissection with blunt instruments and snares.

THE TECHNIQUE

The operation of tonsillectomy for some obscure reason, probably based in antiquity when the tonsil was removed with the fingers without anesthesia, commonly seems to be considered in a category all by itself. Apparently it is immune to the considerations that govern other surgical procedures, such as good exposure, meticulous hemostasis, and careful dissection. Most physicians who remove tonsils are enthusiastic about their operation whenever they are given an opportunity to describe their method. Usually the enthusiasm is for the "slick," "easy," or "quick" method they have adopted. An occasional exception is the competent surgeon who usually admits he finds the operation difficult and time consuming. The 680 patients with tags probably were subjected to a great variety of operative methods, all of which had one thing in common: failure to remove all the tonsil. Certainly a fair test of an operation is that it should accomplish what it intends to do, and such a high incidence of tags following tonsillectomy is *ipso facto* evidence of failure. Irrespective of any consideration of the indications for tonsillectomy and its possible benefits, if the operation is to be done, it is reasonable to expect that the tonsil shall be removed. Most of the justifications for tonsillectomy must be based on complete removal.

It should be legitimate to compare tonsillectomy with the operation for excision of a branchial cleft cyst commonly situated nearby, for example, 1 cm. closer to the skin. They are usually approached with different attitudes however. The difference is that complete excision of the branchial cleft cyst is assumed as a matter of course, whereas portions of the tonsil are usually left in situ. For instance, no surgeon would think of using a snare on a branchial cleft cyst.

Tonsillectomy has certain inherent factors that make it a very difficult operation. The exposure is limited by the size of the mouth, and one cannot improve the exposure by getting an interne to hold a retractor. Illumination can only come from one direction. Hemostasis is difficult because of the small size and depth of the avenue of approach. It is necessary to use long clamps and tie knots at finger tip depth. Moreover the usual position of the patient during operation causes the blood to collect in a pool at the line of dissection. Often the blood runs down the throat causing the patient to gag and cough or arousing apprehension of pulmonary complications.

None of these inherent factors is insurmountable if the surgeon is determined to remove all the tonsil and is willing to take the time and pains to do it by applying the usual standards of any good operation. The position of the patient can be made to facilitate the operation rather than hinder it. For the patient under general anesthetic, the Davis type of mouth gag allows the head to hang inverted over the end of the operating table. The surgeon sits in front of the open mouth. The blood of any uncontrolled bleeding runs down into the nasopharynx and out the nose. The head mirror provides the best illumination, after the surgeon acquires the skill to use it. Intratracheal intubation is sometimes desirable for older patients with stiff necks. One begins the dissection with a sharp knife by outlining the tonsil,

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6. McLaggan, J. D.: A Plea for More Careful Tonsillectomy, Lancet **2**: 1150 (Nov. 22) 1930.

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8. Clark, J. P.: Results in a Series of Cases of Tonsillectomy at the Massachusetts General Hospital, 3 to 4 Years After Operation, Ann. Otol., Rhin. & Laryng. **22**: 421 (June) 1913.

9. Coues, W. P.: The Results of the Clinical Throat Examination of 212 School Children, Boston M. & Sc. J. **162**: 215 (Feb. 17) 1910.

10. Tonsil Stumps, Current Comment, J. A. M. A. **134**: 698 (June 21) 1947.

11. Leshin, N., and Pearlman, S. J.: Are Tonsillar Recurrences Entirely Due to Faulty Operative Technique? Arch. Otolaryng. **13**: 37 (Jan.) 1931.

12. Baum, H. L.: Complications and Sequelae of Tonsil and Adenoid Operations, Ann. Otol., Rhin. & Laryng. **28**: 37 (March) 1919.

including all visible tonsil tissue, especially at the junction of the tonsil and tongue. The tonsil edge is grasped with a tenaculum at the lowest part, which anatomically is designated the superior pole. The dissection proceeds upward and backward by sharp snips with scissors. The line of dissection is always clear, because the blood runs downward and away from it. Spurting arterial bleeding may occasionally jet upward to obscure the line of dissection, and such bleeding is controlled by clamp and tie as in any other operation. The clamp can be applied and left dangling until the dissection is complete. The Sten clamp with the jaws curved at 45 degrees seems most convenient. The tie is made with a 20 in. strand of no. 20 crochet cotton thread. No assistant is necessary. The dissection by scissors is carried completely through to the base of the tongue. No snare is used. There is no reason for one end of the tonsil being dissected any differently from the other end. In fact, the inferior (lingual) pole, which is so often "dissected" by a snare, is more apt to have irregular lobules which require the most careful dissection by scissors for clean removal.

The patient under local anesthetic is seated on an operating table, which is arranged with the foot down and the back up to form a chair. The "knee strap" is tightened across the lap so the patient can lean forward against it. The table is raised to full height. The surgeon sits in front of the patient, who leans forward so the mouth opens downward. The lamp for the head mirror is above the patient, and the surgeon looks upward into the patient's mouth. Blood and saliva drain downward and out of the mouth rather than into the pharynx. After anesthetic infiltration, the dissection is begun by an incision as above described, all the tonsil tissue being outlined. The tenaculum grasps the tonsil at the lower anterior part, at the junction of the anterior pillar and the tongue. The dissection proceeds upward and backward with no opportunity for the blood to collect in a pool at the line of dissection. In this position, each tonsil can usually be removed without the patient's coughing or gagging, except for an occasional spurting artery which may jet across into the pharynx.

RESULTS OF OPERATIVE PROCEDURE

Two hundred sixty-two patients have been operated on in such a manner with a purposeful effort to remove all the tonsil and have been observed six months or more later. The throats of 48 patients (18%) showed some lymphoid tissue in the tonsil area, but in only two of them could the lymphoid tissue be identified as a remnant of tonsil.

SUMMARY

The operation of tonsillectomy as ordinarily performed is considered to be a failure as evidenced by 68% of tonsil tags in a group of 1,000 tonsillectomized patients. Careful dissection, which is facilitated by described technical methods, especially the position of the patient, has been successful in 99% of 262 patients.

The Dalles.

PROBLEMS OF THE GERIATRIC ASTHMATIC AND THEIR CLINICAL MANAGEMENT

Louis Tuft, M.D., Philadelphia

It has been truly said by Stieglitz [1] that old age begins at birth and can be terminated only with death and also that aging is part of living. The recent interest in the study of older or aged people has created the specialty of medicine known as geriatrics, which concerns itself with the physiologic, pathologic, and clinical problems of the elderly. Its need has been stimulated by the remarkable recent increase in the number of older people, brought about largely by the prolonged life span resulting from advances in preventive medicine and therapeutics. As a consequence, many more people now live to 65 or 70 or older and physicians, therefore, must treat many more older people than formerly.

Geriatrics, in a sense, is the converse, or mirror image, of pediatrics and in certain respects is confronted with similar problems. Thus, for example, just as infancy is a critical period in pediatrics, so too is the period of later maturity (between 40 and 60) a critical one for the geriatric patient because of the greater incidence of progressive and degenerative diseases; and since such a patient is an adult growing older, he is also subject to the same diseases and conditions as when younger. Geriatrics therefore embraces many different specialties of medicine: Allergy can and does affect the older person and there is no good reason for assuming that it should not. Hay fever, urticaria, asthma, or atopic dermatitis can begin in people past 40, although less frequently than in younger persons. Asthma, for example, may begin in a person past the age of 75.[2] Recently I saw a 77-year-old patient with typical allergic bronchial asthma which began at the age of 74 and which responded satisfactorily to the usual allergy treatment. For some time, it has been generally considered that allergic conditions rarely begin in senescence or senility and that when they do arise at that time other factors must be operative. This was especially true for asthma. Thus, according to Alexander,[3] asthma is a disease of early decades. He cites the statistics of Coke,[4] who in 1927 found only 20 patients, or 2% of a series of 1,000 asthmatics, to be over 60. The only comparable statistics available at present are those of Rowe and Rowe [5] in 1947. While

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Read before the Allergy Section of the California State Medical Association, San Diego, Calif., May 1, 1950.

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2. This period is designated by Stieglitz as "senility," in contrast to "senescence," which is between 60 and 75.

3. Alexander, H. L.: Bronchial Asthma: Its Diagnosis and Treatment, Philadelphia, Lea & Febiger, 1928.

4. Coke, F.: Review of 1,000 Cases of Asthma, Brit. M. J. 1: 955, 1927.

5. Rowe, A. H., and Rowe, A., Jr.: Bronchial Asthma in Patients over the Age of 55 Years: Diagnosis and Treatment, Ann. Allergy 5: 509, 1947.

these authors did not particularly compute the incidence of asthma in older persons, they mention in their article that of a total of 1,232 private patients there were 72 whose asthma began after 50. This is an incidence of 5.85% and undoubtedly would be higher if only those whose asthma began after 40 were included. Likewise, Fuchs [6] in 1947 reported that, of 200 asthmatics studied, the age of onset at the initial attack was given as past 50 in 27, or 13.5%. This increase in incidence may occur because people are living longer or because physicians are seeing more of them. Despite this, however, some older ideas concerning incidence and etiology in the geriatric asthmatic still prevail, at least as judged by a review of current books and of the sparse literature on geriatrics. It seems worthwhile, therefore, to review this subject, discussing in particular questions of etiology, differential diagnosis, and clinical management. Discussion will concern those patients whose asthma began after midlife and not those who are past 40 and have asthma regardless of when it began. If a patient of 50 has had asthma since he was 20 or 25, the problem of etiology, diagnosis, and management may be quite different from that of a patient of the same age with asthma of only one or two years' duration. Recent articles on this subject by Cueva [7] and by Rowe and Rowe [5] refer to patients past 50 who have asthma regardless of when it started. While their reports are helpful regarding incidence, complications, allergic aspects, etc., they do not differentiate between asthma beginning in the geriatric patient and that beginning before 40.

ETIOLOGY

In the etiology of asthma in the geriatric patient, as contrasted with the younger patient, a difference is observed in the importance of the factor of heredity. Cooke and Vander Veer [8] showed that the significance of hereditary predisposition to asthma decreased progressively after the age of 20. This seems to be verified by clinical experience. Thus, for example, asthma in older people usually is not accompanied with such marked sensitivity to specific allergens as in younger persons, nor is it associated so often with other atopic manifestations such as hay fever, migraine, or eczema. Furthermore, attacks in the geriatric generally are milder, with less tendency toward the explosive acute reactions seen in younger asthmatics, especially in those with strong atopic backgrounds.

Since heredity is not important in predisposing the older person to asthma, the initial attacks may result either from acquired sensitization to inhalants, foods, drugs, or bacterial allergens or from internal causes (endocrine or psychosomatic); the asthma thus provoked may well be the starting point for a new line of allergic offspring in subsequent generations. Undoubtedly older as well as younger persons can be sensitized to inhalant allergens, as, for example, to various occupational dusts. But the extent of exposure required is an undetermined variable which probably depends upon such factors as tissue or cell reactivity, about which relatively little is known. Nevertheless, asthma may be acquired by older persons by contact with environmental allergens; and, as a corollary, exposure to such contacts may be a potent factor in the development of the asthma.

Development of allergy to foods by older persons from continued exposure is less likely than is the case with inhalants. Experience with children has shown that continual ingestion of allergenic foods will sometimes bring about sufficient improvement in tolerance to enable them to eat these foods in average or larger amounts. Therefore, except in exquisitely sensitive patients, many food allergies will be overcome during maturity. Whether allergies to new foods or overindulgence in foods to which they formerly were allergic may be responsible for the food allergy in these persons is difficult to ascertain. Opinion is divided chiefly because most of the evidence is based upon the results of skin tests rather than diet trial. Nevertheless, clinical experience indicates that food allergy may be important in some geriatric asthmatics and its disregard may explain some treatment failures.

Despite these variations, the development of asthma in the geriatric patient and the mechanism of reaction are essentially similar to those of the younger person with one important difference. It has been emphasized unduly by some authors, leading to considerable misconception of the origin of the asthma and often to unnecessary treatment. This is the idea to be found in some textbooks on geriatrics,[9] and also in the publications of some allergists to the effect that asthma developing past midlife is almost always intrinsic or infectious in origin. This thought is so firmly entrenched in the minds of many that if a patient past midlife presents himself with asthma of fairly recent orgin it is assumed at once that the asthma is of infectious or intrinsic origin. For example, Rackemann in a recent article [10] concludes that asthma beginning after age 40 is not often due to allergy but is caused by something which the patient carries with him at all times. He considers infections in the respiratory tract, either primary or secondary, highly important; also, more general disturbances of the whole body, especially emotional or psychosomatic factors, in addition to physical disturbances such as focal infections, malnutrition, or poor general health from any cause. He bases these conclusions, especially as to the psychosomatic factors, upon the results of psychotherapeutic treatment, including reassurance to allay fear, and also upon the fact that "death from asthma can occur from nothing more than emotional strain."

These statements are subject to challenge since they are contrary to the clinical experience of many others. As I pointed out several years ago,[11] the term "intrinsic" or "infectious" asthma often is used when the cause of the asthma is unknown. It should be replaced by the more descriptive term "idiopathic," which describes the situation more accurately. Regardless of these considerations, however, it is difficult to understand why asthma

6. Fuchs, A. M.: Allergy Problems in Elderly Persons, Geriatrics **2**: 235, 1947.

7. Cueva, J.: Asma Bronquial en Geriatria, Rev. mex. de alerg. **1**:5, 1950.

8. Cooke, R. A., and Vander Veer, A., Jr.: Human Sensitization, J. Immunol. **1**: 201, 1916.

9. Thewlis, M. W.: The Care of the Aged (Geriatrics), ed. 3, St. Louis, C. V. Mosby Company, 1941. Boas, E. P.: Treatment of Patient Past Fifty, Chicago, The Year Book Publishers, Inc., 1941.

10. Rackemann, F. M.: Other Factors Besides Allergy in Asthma, J. A. M. A. **142**: 534 (Feb. 25) 1950.

11. Tuft, L.: Bronchial Asthma: A Critical Review, J. Allergy **17**: 27, 1946.

in a patient past 40 must necessarily be regarded as infectious in origin. What it is that makes 40 or 50 or any age the dividing line between infectious and noninfectious asthma is difficult to comprehend.

Hay fever and other forms of allergy can and do begin in persons in the sixth, seventh or eighth decades; why should not asthma of nonbacterial origin? Infections generally occur more frequently in young children than in adults; yet it is agreed that "infectious asthma" is less frequent in children than in older persons. Why, then, assume that asthma beginning in the geriatric necessarily must be infectious?

One explanation may be that positive skin reactions are less frequently seen than in younger persons. This is partly true, if only moderate or marked positive reactions are considered. But since skin reactivity is lessened in older persons, doubtful or slight reactions may be as significant as larger reactions in younger persons. This is even truer of foods, for, as in younger asthmatics, skin reactions may be negative despite the existence of proved clinical food allergy.[5]

Another reason for assuming the infectious nature of asthma in older persons is the presence of evidence of sinus disease (chiefly revealed by x-ray) or of a history of frequent "colds." The fallacy of the former is obvious, since various studies, especially those of Bullen [12] and of Kern and Schenck,[13] have shown that the presence of sinus disease as determined solely by x-ray examination cannot be taken either as a reliable index of infection or as proof that the asthma is infectious. This is true also of the history of repeated "colds." These often actually represent attacks of allergic rhinitis which are mistaken for colds. Many patients with asthma beginning in later life have a history of long-standing recurrent nasal difficulties which upon detailed history and examination prove to be allergic rhinitis. Sometimes the nasal symptoms extend as far back as early maturity or even into childhood and recur only at intervals thereafter or persist in varying degree until the onset of asthma in later years. Not infrequently these patients give histories either of various nasal operations or of recurring nasal polyps, the removal of which provided relief for varying periods. Detailed inquiry into the history often will furnish ample evidence that the asthma was preceded for months or years by an unrecognized allergic seasonal or perennial rhinitis of varying severity. When seasonal, and especially in patients with hay fever of long duration or starting in later life, the initial complicating asthmatic attack may develop in the geriatric period.

It may be concluded, therefore, that there is insufficient evidence to show that asthma in the geriatric patient is necessarily or frequently infectious in origin. Inhalants such as house dust, feathers, pollens, and molds can be important offenders, since many patients with asthma of recent duration improve remarkably after change of environment. Recently a 68-year-old patient with asthma of two years' duration entered the hospital. Within 24 hours after admission, and without treatment, the asthma cleared up spontaneously. He changed residences and remained symptom-free even after his discharge 14 days later, except when he returned for a short stay at his former farm residence. The asthma here, as in other similar patients, undoubtedly was due to environmental factors like those affecting younger asthmatics. Such occurrences prove that asthma due to environmental allergens such as dust or animal danders can affect a person of 70 or more just as it does one of 20. There is little reason for assuming that the asthma is infectious merely because the patient is old.

The importance of allergy to foods already has been emphasized and should be considered in all patients. While opinion as to its incidence may vary, nevertheless, unless an attempt is made to carry out adequate diet trial, it cannot be said categorically that food allergy in the older patient is relatively infrequent. Remarkable benefit sometimes follows the institution of dietary restrictions or trial as in the following case:

CASE 1.—A man, aged 62, with asthma present acutely for 10 months, previously had often been subject to so-called colds and mild coughing attacks occuring mostly in the fall. He had previously been studied in another hospital because of what was believed to be bronchitis and bronchiectasis, both of which were considered upon an infectious basis. He had had a tonsillectomy during his previous hospitalization because of that possibility.

When first seen, the patient was acutely asthmatic and showed some emphysema; otherwise the examination was noncontributory. Immediate hospitalization was refused by the patient, who asked for a delay of a week or two. In the meantime, an allergy diet and also aminophylline by mouth were prescribed Iodides were not used because there was a history of an iodide rash. When he returned a week later, there was striking improvement, with little or no audible wheezing. This persisted throughout the course of his allergy studies and for the two-year period during which he was observed thereafter. He was found to be particularly susceptible to attacks after contact with cooking odors, especially from fish or bacon, or contact with house dust. He was instructed to avoid these and was also given small doses of house dust extract for purposes of desensitization.

This case illustrates the beneficial effects of an allergy diet plus symptomatic measures in a patient with severe asthma previously considered to be upon an "infectious" basis. It might be argued—and with some justification—that the striking relief which followed was not necessarily the result of diet alone, since the patient was receiving aminophylline by mouth at the same time. However, his response was so similar to that of others who were given a diet alone that one is forced to conclude that the dietary restriction or exclusion must be important in such patients. Regardless of these considerations, his striking improvement under allergy treatment, coupled with his clinical reactions to exposure to dust or to cooking odors or cold weather, so closely resembles that of the younger allergic asthmatic as to contradict effectively the original opinion that this was an infectious asthma.

Another etiologic factor considered important in geriatric asthma and believed to contribute to its intrinsic quality is a disturbance of the endocrines, especially in women. Recent work with the pituitary and adrenal hormones has shown that these may have profound influences upon asthma. But we do not know as yet whether disturbances or dysfunction of these glands may be etiologically responsible for asthmatic attacks and, if so, whether this is more prevalent in older persons. The

12. Bullen, S. S.: Incidence of Asthma in 400 Cases of Chronic Sinusitis, J. Allergy **4**: 402, 1933.

13. Kern, R. A., and Schenck, H. P.: Relative Efficiency of Clinical and Roentgenologic Methods for Sinus Diseases Diagnosis, with Observations on Incidence of Sinus Disease: Based on Findings in 200 Asthmatics and 50 So-Called "Normals," Am. J. M. Sc. **178**: 168, 1929.

idea that asthma in women lessens after the menopause is not substantiated by clinical experience; it gets worse in some women instead of better. Proof of the existence of an "intrinsic factor" of endocrine origin as a cause of geriatric asthma therefore still awaits confirmation.

With minor variations, the specific etiologic agents responsible for asthma are essentially the same in the geriatric patient as in younger persons. If this were always remembered, the approach to etiologic diagnosis and to specific treatment in these patients would be the same as though the asthma were affecting a person of 20 instead of 60 or 70.

DIFFERENTIAL DIAGNOSIS

In the differential diagnosis of asthma in the geriatric patient, the age of the patient brings up diagnostic problems which seldom arise in children or younger adults. When an elderly patient presents himself complaining of dyspnea associated with a wheezing respiration, it must first be determined whether the condition is bronchial asthma or a condition resembling it. The dictum of Chevalier Jackson that "all is not asthma that wheezes" applies not only to foreign bodies, especially in children, but also to other obstructive chest lesions such as mediastinal tumors of neoplastic, aneurysmal, or lymphatic origin, which may give rise to similar signs and symptoms. Such tumors usually are distinguishable by the presence of associated laryngeal symptoms (stridor or hoarseness) or of unilateral wheezing, and x-ray evidence of this pathology is distinctive enough to establish the diagnosis with certainty. Pulmonary neoplasms are more difficult to diagnose, especially when associated with typical asthmatic symptoms, because it is not easy to say whether the asthma has been provoked by the neoplastic process or whether the neoplasm exists independently of the asthma. Recently I saw a 76-year-old man with typical symptoms and signs of acute asthma of three weeks' duration and with a proved bronchogenic carcinoma which resulted in his death two months later. Except for the small shadow seen fluoroscopically in the left chest the neoplastic process might have been missed, since the only symptoms and physical signs were those of acute asthma. This emphasizes the necessity for x-ray examination of the chest in all asthmatics, especially older persons, so as not to miss an early pulmonary cancer.

Another condition offering diagnostic difficulties in the geriatric asthmatic is hypertensive cardiovascular disease, particularly so-called cardiac asthma. This term is used to designate a paroxysmal type of wheezing dyspnea, usually nocturnal, which affects those people past midlife with hypertensive cardiovascular disease. It is believed to be due to a sudden relative failure of the left ventricle in a patient whose right ventricle is functionally normal. This sudden failure produces, in turn, acute pulmonary hypertension, secondary intense pulmonary emphysema, and pulmonary edema. Often there is associated precordial pain or oppression of the anginal or coronary type. During the acute attack the condition so closely resembles bronchial asthma that it may be difficult to distinguish the two conditions by examination alone. But because the patient is elderly and has had the asthmatic symptoms for a relatively short time, as well as the presence of hypertension, left-sided cardiac enlargement in varying degrees, cardiac murmurs, and evidence of impairment of conductivity in the electrocardiogram, differential diagnosis between cardiac and bronchial asthma is fairly easy. Unfortunately, however, the history and physical findings are not always typical. My experience and that of our cardiologists indicate that true cardiac asthma is relatively uncommon. Its diagnosis is made more frequently than is warranted, especially in those patients who have nocturnal asthma with mild or moderate degrees of hypertension and no significant cardiac signs. In this situation, it is not always easy to decide whether one is dealing with bronchial asthma in a patient who happens to be hypertensive or whether the asthma is the result of the cardiac lesion. If the asthma has been present for several years and there is definite evidence of allergic sensitization either in the history or from allergy studies, the diagnosis of allergic bronchial asthma is easily established. The allergist frequently sees patients past midlife, especially women, with typical allergic bronchial asthma and a coincident benign essential hypertension. Continued observation of such patients, at least in my experience, shows that the two conditions are entirely unrelated etiologically and should be managed as though they existed independently.

In patients with allergic bronchial asthma of varying duration hypertensive organic cardiac disease or coronary accidents may develop. These have no etiologic relationship to asthma, but they make treatment more difficult, as exemplified in the following case report.

CASE 2.—A man aged 75, was first seen in 1939 with bronchial asthma of three years' duration. The allergic character of the asthma was estabished by a history of attacks of perennial allergic rhinitis preceding the asthma, the presence of urticaria, and specific positive skin test reactions. Despite a blood pressure reading of 170/90, the heart showed no changes at that time. Striking improvement followed allergy treatment, especially after elimination of some very old feather pillows, plus symptomatic treatment and a short course of injections of house dust extract. He remained fairly comfortable for five years without additional or specific treatment until 1944, when at the age of 80 cough, dyspnea, and precordial pain radiating down the left arm developed. Examination showed persistence of hypertension (170/90) and also signs of cardiac insufficiency. Electrocardiograms verified the presence of coronary artery disease. He was treated for the cardiac condition and despite his age improved gradually for a while. He died of cardiac failure three years later at the age of 83.

This case is remarkable from many standpoints, but it illustrates particularly the fact that coronary disease and its aftermaths can affect patients with allergic bronchial asthma without having any etiologic relation to the asthma. Designation of asthma in such a patient as "cardiac" is erroneous and could be avoided if the differentiation between true cardiac asthma and bronchial asthma were kept in mind. This also would eliminate the custom of applying the term "cardiac asthma" to attacks of dyspnea associated with cardiac decompensation or failure. Such usage is incorrect, since neither is the dyspnea periodic nor is it associated with wheezing respiration; it should be designated cardiac dyspnea, not cardiac asthma. When asthma affects patients with such organic cardiac lesions as mitral stenosis, the relationship is likely to be coincidental rather than etiologic. In

such circumstances, the wheezing respiration disappears when decompensation sets in and reappears when compensation is restored.

This brings up for consideration those pulmonary conditions in the geriatric which may simulate asthma. An outstanding example is the chronic bronchitis which, usually associated with chronic emphysema, affects older people. Examination of the chest reveals numerous sibilant and sonorous rales and even a wheezing type of respiration but there is neither paroxysmal dyspnea nor any other of the signs that characterize the acute asthmatic attack. This form often is called asthmatic bronchitis or asthmatoid bronchitis.

Opinions differ as to whether asthmatic bronchitis is a form of asthma due primarily to bacterial sensitivity or, as seems more likely, whether it is entirely secondary to an infection or irritation of the bronchial tubes which causes sufficient bronchial obstruction to produce a mild asthmatic wheeze, perhaps in a predisposed subject. The wheezing usually is secondary to the cough and is more or less continual and not paroxysmal as in true asthma. Patients with asthmatic bronchitis have little or no difficulty in sleeping in a flat position at night, in contrast to the orthopnea of those with paroxysmal or acute attacks of asthma.

Although chronic bronchitis may be due to various causes, a condition frequently responsible for wheezing respiration is pneumonoconiosis, especially the form known as "miners' asthma" (anthracosis). Such patients, after long-continued exposure (more than 12 years) to coal dust, begin to show evidence of bronchial irritation. The frequent more or less continuous attacks of bronchitis are followed by dyspnea upon exertion, with or without wheezing respiration. The latter is the last to appear and is often mild compared with the cough and expectoration. At this stage, emphysema, bronchiectasis, or pulmonary fibrosis are evident. A history of exposure to occupational dust as well as the presence of bronchitis for a long time before the onset of wheezing, together with the physical and x-ray findings, usually are sufficient to differentiate the wheezing of pneumoconiosis from that of true allergic asthma.

Presence of wheezing dyspnea in persons past middle life, usually with moderate or marked emphysema; predominance of protracted cough accompanied with little or no dyspnea in spite of signs of wheezing in the chest; presence of mucopurulent sputum, and absence of any evidence of allergy either in the history or by special examination are all of differential diagnostic importance. They serve also to distinguish asthmatic bronchitis from the bronchitis present during or at the end of an attack of allergic asthma.

Although these conditions always have been considered as instances of primary chronic bronchitis with secondary asthmatic symptoms or as nonallergic asthmas, it is not unlikely that the asthmatic wheezing associated at times with certain obstructive or nonobstructive chest conditions (e. g., neoplasm, "cardiac asthma," etc.) is not due to any direct influence of the original causative lesion. These patients may possess in their bronchi what might be called an asthmogenic tendency, which lies dormant perhaps for years until activated by a lesion such as neoplasm, tuberculosis, cardiac disease, or pneumonoconiosis. These processes then act as triggers to set off the dormant asthmatic tendency. It seems difficult otherwise to explain why, of the many patients with pulmonary neoplasm or hypertensive coronary disease, relatively few have asthmatic wheezing as a prominent symptom or why, of the many miners with chronic bronchitis and emphysema, relatively few contract miners' asthma. The development of asthma in such patients must depend upon some factor other than the associated pulmonary or cardiac lesion. Recently, for example, I saw a mine inspector, aged 50, with asthma of three years' duration whose father had miners' asthma. X-ray examination showed some associated emphysema but little fibrosis. He has responded very well thus far to allergy treatment similar to that given younger asthmatics. Fortunately he did not have much exposure to coal dust; otherwise, he surely would have had miners' asthma as his father did. Tissue predisposition must be important in such patients, despite an apparently negative atopic family history.

Differential diagnosis is of primary importance in the geriatric asthmatic. Detailed history and thorough physical examination, especially of the lungs and cardiovascular system, are paramount. Competent x-ray examination of the entire chest cavity should be a routine procedure in all these patients and, whenever necessary, such special cardiologic examinations as electrocardiography, determination of circulation time, etc. These should make it possible to arrive at the proper diagnosis and to distinguish true allergic bronchial asthma from the nonallergic asthmas, including so-called asthmatic bronchitis and cardiac asthma. The diagnosis is established more definitely by the presence of increased numbers of eosinophils in the blood and the nasal and bronchial secretions; a history of associated allergic conditions such as allergic rhinitis, migraine, urticaria, etc. and specific positive skin reactions.

TREATMENT

When the diagnosis of allergic asthma has been made, the patient should be managed as though he were 20 or 30 instead of 50 or 60. Attempts should be made to determine the specific allergen or allergens by history, allergic tests, and clinical diet trial. It should be kept in mind that the skin of the geriatric is less reactive than that of younger persons and that positive reactions of even doubtful or slight degree should be considered important until proved otherwise. Diet trial should be carried out whenever food allergy is suspected. But it must be remembered that the nutritional state in the old as in the very young can be greatly impaired by severe or prolonged dietary restriction unless proper precautions are observed. Provision should be made for adequate caloric intake and for supplemental vitamins and minerals. Diet trial often will improve the condition of such patients, especially those previously thought to have "infectious" or "intrinsic" asthma.

As stated above, there is no proof that infection, especially in the paranasal sinuses, is the most important cause of asthma in the geriatric patient. Care is therefore needed in choosing treatment for these patients, even when there is x-ray evidence of sinus infection. I once had an elderly asthmatic patient who, though his condition was much improved with conservative allergy treat-

ment, was discouraged by the persistence of mild attacks. A radical sinus operation was advised by his otolaryngologist, who had treated him for a long time for nasal difficulties. While I was not at all certain it was essential, I made no effort to interfere. The patient died from a complicating pneumonia a week after the operation. Since then, unusual caution has been exercised with these patients; furthermore, such procedures are seldom advocated except when purulent infection that does not respond to ordinary drainage and treatment exists. As nearly as can be judged from the reports in the literature,[14] as well as from subsequent clinical experience, the results of conservative treatment are just as good as, if not better than, those of radical sinus surgery. Conservatism in treatment is the keynote of success in all geriatric asthmatics.

The treatment of asthma in the aged is generally the same as in younger persons—elimination or avoidance of the offending allergens, or desensitization when the former is not possible. As in young children, due care should be exercised in administering extracts of dust, pollens, molds, or bacterial vaccines to avoid constitutional reactions, which may upset older patients more than younger persons. We sometimes see older asthmatics who previously received large doses of vaccines or dust extracts and who seem to get well even faster than younger and previously untreated asthmatics with only simple symptomatic treatment. The improvement in these patients probably is due largely to discontinuance of the injections of extracts in a dosage which did more harm than good. When in doubt, saline injections should be substituted for several weeks. If improvement follows, it is certain that the allergenic extract was causing reactions. The dosage then either is modified accordingly or the injections are discontinued entirely. Except for these precautions, the specific desensitization procedures are the same in the geriatric as in younger patients.

More care should be exercised, however, in the use of nonspecific methods of desensitization, especially in relation to fever therapy provoked by intravenous injections of typhoid or colon bacilli. The vascular systems of the aged naturally will not withstand the shock of sudden reactions as easily as those of younger persons; unless these injections are given cautiously, severe untoward or even fatal reactions may follow. When in doubt, it is best to avoid them.

Regardless of cause, drug therapy for symptomatic relief is an essential part of treatment in the geriatric asthmatic. As a rule, the same drugs are employed for any asthmatic patient, regardless of age. Of all drugs used, potassium iodide is one of the mainstays in treatment. Given in doses of 10 or more grains (0.60 Gm. or more) three times daily it seems to have a particularly beneficial effect on the elderly asthmatic. Whether this is due alone to its liquefying effect on the sputum or to some peculiar alterative effect on the tissues of the aged as well is not known; nevertheless, it is very helpful during the subacute or chronic stages. Ephedrine or one of its synthetic substitutes may be given at the same time, but more care should be exercised in their use, especially in the older male patient with prostatic hypertrophy. Ephedrine has produced in some patients such vesical tenesmus that prostatic surgery was considered; when the symptoms disappeared afte the medication was discontinued, the urologist realized that the symptoms were being provoked by the ephedrine and operation was avoided. Therefore, if the ephedrine drugs are to be used in older patients, smaller doses should be employed than in younger adults.

Treatment of the acute asthmatic attack in the aged generally is like that in any other asthmatic. Epinephrine by injection or aerosol still is the mainstay, with aminophylline by mouth, rectum, or intravenous injection as a substitute or supplement when epinephrine either is ineffectual or cannot be used. The question whether to use epinephrine or aminophylline for the relief of the acute attack often arises in the geriatric because of the likelihood of hypertension. If the hypertension is benign or essential, epinephrine in small doses can be used without fear to relieve acute asthma. Practitioners often are afraid to use it in such circumstances because they feel the epinephrine might increase the blood pressure. In my experience, however, it does the opposite. The relief of the asthma afforded by epinephrine usually reduces the blood pressure, which had been elevated by the vascular strain induced by acute asthma. One should not hesitate, therefore, to use small doses of epinephrine for relief of acute attacks unless the patient shows definite evidence of organic cardiac disease, especially involving the coronary artery. In those circumstances, the drug of choice should be aminophylline, given either orally or rectally, or, in acute attacks, by intravenous injection. In the latter instance, it should be administered slowly and cautiously. The fatal reactions after intravenous injections of aminophylline reported by Merrill [15] in 1943 occurred mostly in asthmatics with severe organic cardiovascular disease and might have been due either to rapid administration or to the fact that these patients were in extremis. In such instances, aminophylline rectally should be tried first and, if ineffective, may then be given intravenously in smaller dosage. This is also the only instance in which morphine may be given justifiably to an asthmatic patient. Since morphine depresses the respiratory system, its use in other asthmatics is contraindicated. But if a diagnosis of cardiac asthma has been established, it then can be given with benefit. If in doubt, meperidine (demerol®) hydrochloride may be somewhat safer, although it too should not be used unless essential. The aged, like children, do not tolerate sedatives such as barbiturates or morphine drugs as well as do younger persons; hence more care is needed in their administration.

Two other remedies sometimes given to the geriatric patient, whether asthmatic or not, are digitalis and alcohol. The indications for their use are not always clear, nor is there certanty as to their value. Unless cardiac insufficiency exists, the efficacy of digitalis is doubtful. Alcoholic drinks are believed to be tonic and supportive to the older person. Unless there is clinical evidence that their imbibition aggravates the asthma, they may be useful adjuncts. Relief of mild attacks might be obtained by their moderate use.

14. Guerrant, J. L.; McCausland, A., and Swineford, O., Jr.: Asthma with Sinus Disease: Comparison of Radical Sinus Surgery and Conservative Treatment, J. Allergy **21**: 187, 1950.

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EMPHYSEMA

The most important complication of asthma in the geriatric is emphysema. This will be present in varying degrees depending upon the character and chronicity of the asthma. When asthma begins after 40 and recurs in the form of acute attacks, little emphysema probably will result. But if asthma becomes chronic and lasts for five years or more, emphysema of increasing severity will follow. It is especially troublesome in patients with asthmatic bronchitis in whom it is responsible for the annoying dyspnea accompanying the cough. Emphysema in these patients is due to more or less continuous overdistention of the pulmonary alveoli, leading to loss of elasticity of the connective tissue and its eventual destruction; it is much like an overstretched rubber band that fails to recoil. Increasing emphysema results in increasing reduction of pulmonary function, indicated by vital capacity or by bronchospirometric tests and clinically by increasing dyspnea. Little can be done to alter the status of a patient with permanent tissue changes indicated by a fixed chest wall. Nevertheless, some relief may be afforded by symptomatic measures to counteract the asthmatic wheezing, the cough, and the associated bronchial infection. In addition, some patients will derive benefit from the abdominal belt used in emphysema and perhaps from breathing exercises designed to improve diaphragmatic excursions or from a positive pressure-oxygen apparatus.

CONCLUSIONS

Asthma in geriatric patients usually has the same general characteristics, etiology, and clinical features as in younger people. However, the geriatric asthmatic differs in certain respects from the younger adult asthmatic, as does the asthmatic child. He also presents different diagnostic problems, especially those in connection with intrathoracic and cardiovascular conditions which require special attention and methods of diagnosis. Treatment, although generally the same in principle, also brings up special considerations and problems, as, for example, in the use of epinephrine, ephedrine, and aminophylline, which are of less moment in younger individuals. Conservatism is the keynote to successful treatment of the geriatric asthmatic. Properly utilized, these measures will enable the practitioner to attain the ideal goal of successful treatment in the geriatric patient, which should be to add life to his years as well as years to his life.

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PHYSIOLOGICAL BASIS FOR PRESERVATION OF FECAL CONTINENCE AFTER RESECTION OF RECTUM

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Preservation of the anal sphincters in resection of the rectum can have but one aim, the retention of fecal continence. If this aim is to be realized, the mechanism by which fecal continence is normally maintained should be understood. The experimental work of McVay,[1] Gilchrist and David,[2] Glover and Waugh [3] and others [4] indicates that lymphatic metastasis from cancer of the rectum does not take place in a retrograde or distal direction until the proximal nodes lying in the mesosigmoid are largely blocked by malignant cells and the lymph stream thereby forced into collateral channels. On the basis of this work some surgeons have concluded that it is both safe and desirable to preserve the anal sphincters when resecting the rectum for malignancy. Other surgeons have violently disagreed with this view on the basis that the less radical, sphincter-preserving operation will result in fewer cures. The purpose of this paper is not to enter into a discussion of this controversial point but rather to summarize the physiological basis on which fecal continence is maintained. It seems obvious that if the anal sphincter is preserved, but its function lost, little, or nothing has been accomplished.

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TYPES OF FECAL CONTINENCE

Fecal continence may be defined as the ability to retain the contents of the colon until their expulsion is convenient. To understand how this is accomplished in normal persons, it is of the greatest importance to realize that fecal continence may be subdivided into reservoir continence (previously termed colonic continence) and sphincteric continence.

Reservoir Continence.—The left colon is fundamentally a reservoir organ and, like other smooth muscle reservoirs, automatically and without sensation adapts itself, up to a given point, to the bulk of its contents. When this bulk reaches a certain magnitude, peristalsis begins and the fecal mass is ejected. This ejection is beyond voluntary control. However, it is to be noted that feces are retained in the left colon for appreciable periods of time, 24 to 48 hr., and that freedom from colonic emptying over such periods constitutes a type of continence that has nothing to do with anal sphincters. Indeed, if the rectum and anal sphincters are entirely removed and a sigmoid colostomy established through the abdominal wall, this reservoir function of the colon can, by attention to diet and colonic irrigations at regular intervals, be utilized to produce a type of continence which experience has shown to be surprisingly satisfactory. For convenience, this has been termed reservoir continence, and it cannot be too strongly emphasized that it is retained whenever all or part of the left colon is preserved. Thus, if a sigmoid colostomy is established on the perineum and encircled by anal sphincters which

are entirely functionless, reservoir continence can be developed by the same measures used in abdominal colostomy and the resulting continence mistakenly attributed to the retained sphincters. There is reason to believe that, in a number of reported series in which the functional value of retained sphincters has been evaluated, exactly this mistake has been made.

Sphincteric Continence.—The term sphincteric continence refers to the active contraction of the striated muscle of the external anal sphincter, which thereby resists the propulsive force of colonic peristalsis. Denny-Brown and Robertson [5] have shown that there is no damping, or inhibitory, effect on colonic peristalsis when the sphincter is voluntarily contracted. The sphincter therefore acts by mechanically blocking the progress of the fecal mass, and the force of sphincteric contraction must exceed the propulsive force of colonic peristalsis to be effective.

It is worthy of emphasis that normal persons have both reservoir continence and sphincteric continence; that is, a smooth muscle reservoir lies above normally functioning sphincters. This is an important consideration when the ileum, which normally is not a reservoir organ, is brought down to the anal sphincters after total colectomy for such diseases as ulcerative colitis and multiple polyposis.

THE ANAL SPHINCTERS

Textbooks of anatomy commonly describe two anal sphincters: the internal and the external. The internal sphincter consists of a condensation of the circular smooth muscle fibers of the rectum. Like smooth muscle elsewhere in the body, it is under the control of the autonomic nervous system and, as will be demonstrated, takes part merely in the peristaltic activity of the smooth muscle of the rest of the rectum and has nothing to do with sphincteric continence. Thus, the statement, repeatedly noted in surgical literature, that division of the internal sphincter causes incontinence is misleading. Surgical division of the internal sphincter is necessarily accompanied by complete division of the external sphincter, and it is this latter maneuver which results in loss of sphincteric continence.

The external anal sphincter consists of striated muscle, is innervated by somatic nerves, and, therefore, is under the control of the voluntary nervous system. It is commonly assumed that this sphincter functions as a simple purse string. That such is not the case has been demonstrated by experimental studies which have been previously reported.[6] These studies point the way to successful methods for the preservation of fecal continence and have been amply confirmed by clinical observation.

SUMMARY OF EXPERIMENTAL STUDIES

Like striated muscle in other parts of the body, the external anal sphincter fatigues quickly. This fact can be readily confirmed by introspection and has been demonstrated experimentally by placing a balloon, connected for kymographic recording, within the anal canal. After the temporary spasm caused by the introduction of the balloon has subsided and the basic tonus of the sphincter has been noted, the subject is requested to contract the sphincter with all possible force and for as long a time as possible. An analysis of graphs obtained by this method indicates that maximal contraction is sustained for only a few seconds and, even with continued effort, is followed by a steady and progressive fall of sphincter tone until the base line is reached. In a series of normal persons, the average maximal contraction was 44 cm. of water and the average length of time during which any contraction was possible was 53 sec.

The fact that the sphincter fatigues so readily makes it evident that some mechanism is required to correlate the activity of the sphincter with the peristaltic activity of the colon. The fact that such a correlation exists has been demonstrated by filling the colon to the limit of toleration with saline solution by means of an enema. This stimulates peristalsis, and the increases in intrarectal pressure brought on by peristalic waves have been recorded kymographically. Balloon recordings of sphincter tone made simultaneously demonstrate that with each peristaltic wave the increased pressure exerted against the outlet is countered by an appropriate degree of contraction of the anal sphincter. As the peristaltic wave recedes and the pressure against the sphincter falls, relaxation of the sphincter takes place, only sufficient tone being maintained at any given moment to counter effectively the peristaltic thrust. Since peristaltic waves ordinarily last less than one minute and since the peak of each wave lasts only a few seconds, it is evident that maximal contraction of the sphincter is required for only a few seconds at a time and that its endurance is sufficient for the demands ordinarily put upon it.

The following facts were repeatedly and consistently demonstrated in normal adults by the application of a method which made it possible to record simultaneously and independently on a kymograph the tone of each of the two sphincters:

1. Stimulation of the rectum by distending a balloon within its lumen caused a sustained fall of internal sphincter tone. This fall was proportional to the degree of distention of the rectal balloon and was sustained until the balloon was deflated. Thus, the internal sphincter evidently plays no part in sphincteric continence but rather, by its relaxation during rectal stimulation, prepares the way for defecation. This rectal stimulation also caused a sharp rise of external sphincter tone which was of short duration. The greater the stimulus applied to the rectum (i. e., the larger the balloon) the greater was the force of the corresponding contraction, but the duration of the contraction was not increased. Thus, as the intrarectal pressure was increased by enlargement of the balloon, the external sphincter contracted only sufficiently to maintain continence. As soon as the intrarectal pressure was diminished by relaxation of the smooth muscle of the rectum, sphincteric contraction was no longer needed and fell rapidly to its original level.

2. Stimulation of the sigmoid colon by the same method caused no change in the tone of either the internal or the external sphincter.

3. When the balloon was placed high in the rectum, near the rectosigmoid junction, the stimulus resulting from its distension caused the characteristic responses of both the internal and external sphincters; but the responses were feeble. When a similar stimulus was applied to the midrectum, the magnitude of the responses was greater. When the stimulus was applied to the distal portion of the rectum, still greater sphincter responses

5. Denny-Brown, D., and Robertson, E. G.: Brain **58**: 256-310, 1935.
6. Gaston, E. A.: Surg., Gynec. & Obst. **87**: 280-290 and 669-678, 1948.

were obtained. Thus, the farther distally in the rectum the stimulus was applied, the greater were the sphincter responses.

4. Stimulation of the rectum in a patient with complete transection of the spinal cord produced a normal relaxation response of the internal sphincter but no corresponding contraction response of the external sphincter.

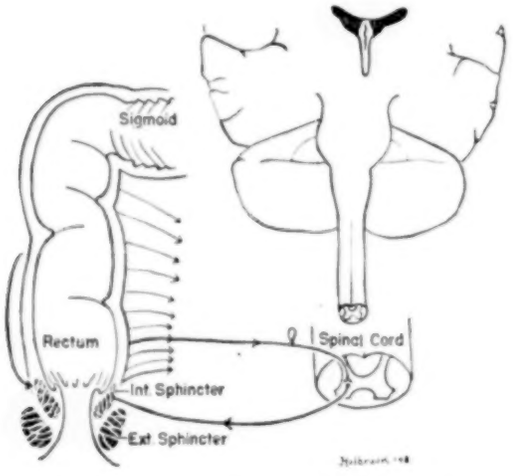

Fig. 1.—The reflex connections between the rectum and the internal sphincter.

COMMENT

The experimental findings indicate that nervous reflexes exist between the rectum and the internal sphincter and between the rectum and the external sphincter and that there is no such reflex connection between the sigmoid colon and the sphincters. The receptor units which initiate these reflexes are sparse per unit area in the upper rectum and become progressively more numerous per unit area in the lower rectum. Because the internal sphincter reflex is retained in complete transection of the spinal cord, it is evident that higher cerebral centers are not necessary to its characteristic relaxation response (fig. 1). This point is of only academic interest since, as has been shown, the internal sphincter plays no part in sphincteric continence. However, the external sphincter reflex is destroyed when the spinal cord has been transected; and cerebral connections must, therefore, be required for its completion (fig. 2). The reflex arc of the external sphincter may also be broken by division of the peripheral motor nerves which supply the external sphincter. It is conceivable that such an accident could occur during resections of the rectum since the inferior hemorrhoidal nerves to the external sphincter are closely applied to the inferior surfaces of the levator ani muscles, which must be divided when the lower part of the rectum is freed.

Most pertinent to this discussion, however, is the fact that the reflex arc can be broken by removal of the afferent nerves which originate only in the rectum. When all of the rectum is removed, the receptor units of the rectosphincteric reflexes are also removed, and the mechanism by which the external sphincter is warned as to when and to what extent its services are required is destroyed. It is therefore evident that the rectum is as essential to sphincteric continence as is the sphincter itself.

It has been possible to confirm the importance of the afferent portion of the reflex arc by investigation of a woman patient who had the entire rectum removed and the sigmoid colon pulled down to take its place. The external sphincter and its motor nerve supply were preserved intact, for it can be voluntarily contracted and has normal strength and a normal fatigue curve. Sphincteric continence does not exist, however, because bowel content escapes before there is any warning that its escape is imminent, and balloon studies show no reflex response of the sphincter when the "new rectum" is stimulated. It has been necessary to place this patient on a colostomy regimen consisting of attention to diet and colonic irrigations every second day. On this regimen reservoir continence, exactly as in a patient with abdominal colostomy, has been developed to the point where she is able to carry on her usual activities. From the standpoint of fecal continence, the retained sphincter is of no functional value.

Since resection of all of the rectum leads to loss of sphincteric continence, the question arises as to how much rectum must be retained in order to preserve satisfactory sphincteric function. From the study of a number of patients in whom varying amounts of rectum have been preserved it would appear that approximately the distal fourth of the rectum is required for this purpose. In this respect it should be emphasized that the rectum varies in length in different persons. It is longer in women than in men and longer in tall people than in short people. For this reason it is probably best to express the amount of rectum that is required for the

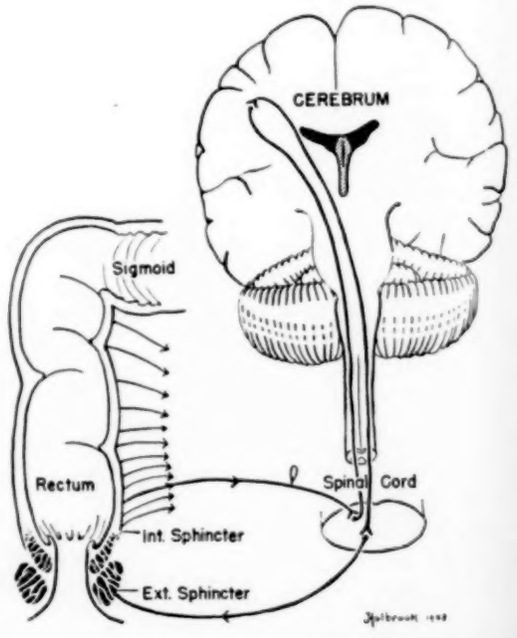

Fig. 2.—The reflex connections between the rectum and the external sphincter. Cerebral connections are required for the completion of this reflex.

preservation of sphincteric continence in terms of the initial length of the rectum, as measured with the sigmoidoscope

Because the sensory nerve supply of the distal colon and rectum parallels that already described for the

afferent portion of the rectosphincteric reflex arc, it is possible to determine clinically when the reflex is intact. If a balloon is distended in the sigmoid colon, sensation is produced in the lower abdomen, usually in the midline but often in the left lower quadrant, which is interpreted as a cramp and not as a desire to defecate. When the balloon is pulled down to a level just below the rectosigmoid junction, a similar degree of distension causes in the sacral or perineal region a sensation which is interpreted as a mild desire to defecate. The lower in the rectum the balloon is placed, the greater is the sacral or perineal sensation resulting from an equal degree of distension. Therefore, if the patient who has had a portion of the rectum removed has a sensation located in or near the perineum which approximates that to which he is normally accustomed when a bowel movement is imminent, and if he is able to contract voluntarily the external sphincter in a normal fashion, both the afferent and efferent portions of the reflex are intact, and true sphincteric continence exists.

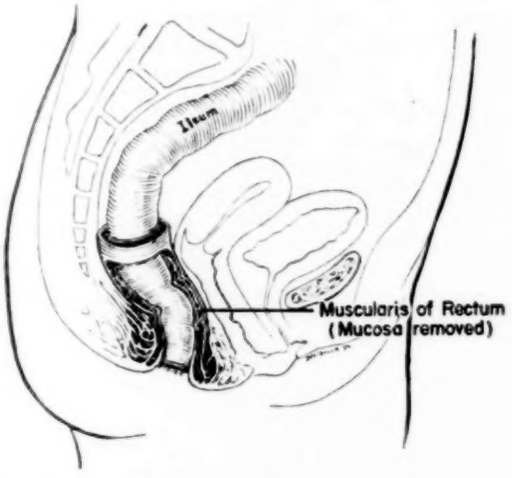

Fig. 3.—Diagram of Ravitch operation. Since the muscular wall of the rectum is retained, the afferent portion of the rectosphincteric reflex is preserved.

Since reservoir continence alone can be developed to such an extent that an individual is able to resume his social obligations without embarrassment, it has been of some interest to observe a patient who has only sphincteric continence. This concerns a woman, aged 43, who suffered recurrent bouts of ulcerative colitis for a period of 11 years. A terminal ileostomy was done in June, 1947, because of a severe exacerbation of the disease, and was followed by dramatic improvement in her general condition. On March 4, 1949, approximately two years later, a total colectomy and anal ileostomy were performed using the technique described by Ravitch.[7] The entire colon and all but the distal 7 cm. of the rectum were removed. The mucosa was removed from the retained distal segment of rectum and the terminal ileum brought through this muscular cuff and sutured in place (fig. 3). Thus, the sphincter and its motor nerve supply were not disturbed and the afferent portion of the rectosphincteric reflex arc was preserved in the retained muscularis of the rectum. The postoperative convalescence was complicated by pelvic sepsis, which required the restablishment of an abdominal ileostomy and local drainage. Eventually the sepsis cleared and healing was complete. The abdominal ileostomy was closed in November, 1949. Approximately five months later the patient had a perfectly normal anal sphincter and sufficient rectal muscularis with its afferent nerve supply so that a normal rectosphincteric reflex remained. The desire to defecate was manifested by a normal sensation located in the perineum. While she was able to retain the contents of the ileum by sphincteric contraction in a normal fashion, she was able to do so for only short periods of time and went to stool at least once an hour throughout the day and night. It seems evident that the poor result in this case is due to the fact the terminal ileum is not normally a reservoir organ and, until that time, had failed to take over the function of a reservoir. Thus, sphincteric continence exists without associated reservoir continence and the functional result is much less satisfactory than when the reverse is true.[8]

SUMMARY AND CONCLUSIONS

Fecal continence can be subdivided into reservoir continence and sphincteric continence. Both reservoir continence and sphincteric continence must be retained if normal fecal control is to be preserved. Reservoir continence alone is of greater practical value than sphincteric continence alone.

The preservation of reservoir continence requires the retention of some portion of the left colon to act as a reservoir organ. The ileum is often unsatisfactory for this purpose.

Sphincteric continence depends upon a nervous reflex, the afferent fibers of which arise in the rectum, the efferent fibers terminating in the external anal sphincter. The rectum is therefore as much a part of the sphincter mechanism as is the sphincter itself. If all of the rectum is removed, sphincteric continence is lost because this reflex arc is broken by removal of the afferent nerves.

If sphincteric continence is to be preserved in rectal resections, it is necessary to retain not only the external anal sphincter but also some portion of the immediately superjacent rectum. Probably the distal fourth of the rectum is sufficient for this purpose.

190 Concord St.

7. Ravitch, M. M.: Surgery **24**: 170-179, 1948.

8. Apparently the ileum is sometimes able to take over the function of a reservoir, for Ravitch has reported good functional results in several of his cases. However, a number of other surgeons have found the operation unsatisfactory because of the intolerable frequency of stools.

The Cortical Steroids.—The measurement of 17-ketosteroids in the urine is of no value in assessing the rate of secretion of cortical hormone in man. Considerable confusion has arisen in the clinical literature due to misinterpretation of the significance of urinary 17-ketosteroids. There is a distinct difference between the pattern of urinary steroids in hypercorticism induced by ACTH administration and in adrenal hyperactivity induced by stress. In hypercorticism both corticoids and 17-ketosteroids are excreted at a rate greater than normal; in adrenal hyperactivity associated with stress the corticoids are increased but the 17-ketosteroids are usually decreased. The evidence indicates that the metabolic pathways concerned with the "utilization" and/or degradation of the cortical steroids undergo qualitative and possibly quantitative changes under the influence of stress.—George Sayers, Regulation of the Secretory Activity of the Adrenal Cortex, *The American Journal of Medicine*, May, 1951.

NONSPECIFIC ULCER OF THE SMALL INTESTINE

REPORT OF FIVE CASES

Patrick C. Shea Jr., M.D., Atlanta

Nonspecific ulcer of the small intestine is one of the rare and particularly enigmatic diseases of the abdomen. Little is known of this intestinal lesion. The clinical picture is vague and confusing, making preoperative diagnosis almost an impossibility. The etiology is unknown. The histologic characteristics are a matter of record, but they give little indication of the pathogenesis of the lesion.

Excellent papers have appeared in the literature tabulating the cases which have been reported and summarizing what is known of the disease.[1] This type of ulcer was first described in 1805 by Matthew Baillie; yet in 1947, when Evert, Black, and Dockerty [1a] at the Mayo Clinic made a study of the literature, they found a total of only 130 cases, including 14 from their own clinic. The purpose of this paper is to report five cases which were seen in the past three years in a city hospital which admits a yearly average of 22,000 patients.

REPORT OF CASES

CASE 1.—W. D., a Negro man aged 39, had had persistent abdominal distention for 12 days, accompanied with intermittent colicky abdominal pain, nausea, and vomiting. The patient was acutely ill and had a temperature of 100.2 F. on admission. The abdomen was protuberant and hyperresonant to percussion. There was generalized tenderness referred to the right lower quadrant. Peristalsis was present but hypoactive. Examination of the rectum revealed moderate tenderness high on the right. Roentgenographic examination of the abdomen revealed severe distention of the entire small intestine.

Laparotomy was performed, and about 500 cc. of pinkish-tan peritoneal fluid was encountered. The distal ileum and the cecum were collapsed. The proximal small intestine was distended with fluid. In the midileum an area approximately 30 cm. in length was violaceous in color; multiple perforations were present and were loosely sealed with overlapped segments of dilated intestine. The intestine was emptied of its contents by aspiration through one of the perforations. The diseased segment was resected and an end-to-end anastomosis was established.

Postoperative examination of the small intestine by contrast roentgenogram, typhoid and brucellosis agglutination tests, and cultures of the urine and stool for typhoid bacilli were all negative. The patient was seen three months later and was found to be asymptomatic.

Histologic Examination.—Near one end of the segment of ileum, there was a rent about 5 cm. in length that extended completely through the intestinal wall and had rather smooth edges. The serosal surface was shiny and mottled deep purplish-red. The intestine was edematous as was the mesentery attached to it. All layers of the intestinal wall were involved in a chronic inflammatory reaction. There were multiple areas of dissolution of the mucosa with ulceration, and healed lesions were present. The medium-sized veins and a few arteries in the wall showed segmental destruction of the vessel wall, associated with a developing thrombosis.

CASE 2.—G. B., a Negro man aged 48, was admitted to the hospital for regulation of mild cardiac decompensation. There were no abdominal complaints, and the history concerning gastrointestinal disease was insignificant. On the fourth day mild pain developed in the left lower quadrant, accompanied with nausea. The pain persisted during the ensuing 24 hours and became increasingly severe and colicky.

The white blood cell count had risen from 6,700 on admission to 13,600; the nonprotein nitrogen blood level was 61 mg. per 100 cc. Blood pressure was 190 systolic and 140 diastolic; temperature was 101.8 F. The heart was well compensated but fibrillating slowly.

Examination of the abdomen revealed generalized tenderness referred to the left lower quadrant. Peristalsis was present but hypoactive. The liver was enlarged but nontender. Examination of the rectum was noncontributory.

Laparotomy was performed. There was no visible free fluid when the peritoneal cavity was opened. An area of thickened, edematous jejunum was noted approximately 45 cm. from the ligament of Treitz. In the middle of this area there was a patch 2.5 by 1 cm. in which the serosa was yellow and mottled with red. Palpation at that point demonstrated thinning of the intestinal wall. The entire thickened area, including margins of 5 cm. of normal intestine, was resected, and an end-to-end anastomosis was established.

The patient was examined two and one-half months after the operation and was found to be asymptomatic.

Histologic Examination.—The specimen was a segment of jejunum measuring 17 cm. in length. The mucosal surface was interrupted by a 3 by 1.5 cm. yellowish-green area of ulceration. The mucosa surrounding this area was fiery red. An ulcer involved a portion of the muscularis, the submucosa, and the mucosa. An acute inflammatory process extended through the wall of the intestine. The vessels appeared dilated and congested; several contained fibrin thrombi, and some of the vessel walls showed infiltration by polymorphonucelar leukocytes. The serosal surface showed acute inflammation and the presence of fibrin.

CASE 3.—C. B., a boy aged 19, had had sudden onset of lower abdominal pain, colicky in nature and accompanied with one episode of vomiting, about six hours previous to admission. Several months previously an appendectomy had been performed for a ruptured appendix, later followed by division of adhesions to relieve intestinal obstruction. There had been no other complaints.

The patient's temperature was 100.2 F. on admission. There was moderate rigidity of the abdomen with extreme rebound tenderness over the umbilicus and in both lower quadrants. Peristalsis was absent. Examination of the rectum revealed tenderness high on the right and in the midline. Roentgenographic examination of the abdomen disclosed nothing of significance.

Laparotomy was performed by Dr. Lewis Sharman. There was a small amount of cloudy peritoneal fluid. Two perforations were noted approximately 25 cm. proximal to the ileocecal valve: One was approximately 1 cm. in diameter; the other, 3 mm. In the area of the perforations, the intestine was edematous and thickened; distally, the lumen was narrowed but not occluded. There was no evidence of mechanical obstruction. The involved area of the ileum was resected, and an end-to-end anastomosis established.

Contrast roentgenographic examination of the abdomen one month after the operation revealed a normal ileum. The patient was seen at intervals for three years; the only complaint he had was occasional cramping abdominal pain. Results of roentgenographic examination of the gastrointestinal tract and physical examination at his last visit were normal.

Histologic Examination.—The specimen was 5.5 cm. of ileum, measuring in circumference 7.5 cm. at one end, 3 cm. in the middle, and 4.5 cm. at the distal end. Just proximal to the constricted area was a perforation 1.5 cm. in diameter with ragged edges. The surrounding serosal surface was granular and cov-

From the Whitehead Department of Surgery, Emory University, and the Grady Memorial Hospital.

1. (*a*) Evert, J. A.; Black, B. M., and Dockerty, M.: Primary Nonspecific Ulcers of the Small Intestine, Surgery **23**: 185, 1948. (*b*) Morrin., F. J.: Spontaneous Perforation of Primary Jejunal Ulcers, Irish J. M. Sc. (ser. 6) 198, 1931. (*c*) Ebeling, W. W.: Primary Jejunal Ulcer, Ann. Surg. **97**: 857, 1933.

ered by an exudate composed of fibrin and neutrophils. The mucosal surface showed regenerating epithelium with hyperplasia, but there was no evidence of malignancy. The muscular layer terminated a short distance from the perforation by gradually thinning. The appearance of the muscular coat suggested that the perforation occurred at the site of a diverticulum.

CASE 4.—M. D., a man aged 80, had a history of indigestion for five days prior to admission. He denied previous intestinal disorders. A local physician reported that two days before admission, examination had revealed a hernia which was easily reducible; the abdomen had been soft and nontender, and peristalsis had been active.

Examination on admission revealed an acutely ill patient with a temperature of 102 F. The odor of his breath was slightly uremic. Rales were present in both lung bases. The abdomen was slightly distended and exquisitely tender, with rebound tenderness in the entire lower abdomen. Peristalsis was absent. There was a cystic, nontender mass in the left inguinal region. Examination of the rectum revealed nothing significant. A roentgenogram of the abdomen disclosed no abnormality and a chest film revealed no evidence of pulmonary disease. The white blood cell count was 13,500; the nonprotein nitrogen level was 50 mg. per 100 cc.

Herniotomy was performed, but no incarcerated intestine was found. Therefore, an exploratory laparotomy was performed by Dr. Wilbur Scott. In the proximal 30 cm. of the jejunum there were matted and adherent intestinal loops bound one to another with fine fibrinous and fibrous adhesions; in the midportion of this area was a hemorrhagic, friable segment of intestine 15 cm. in length, in the center of which a perforated ulcer was present. Biopsy was performed, and layer transverse closure of the intestine was used.

Postoperatively the patient was moderately disoriented for 48 hours and then gradually improved. On the fifth day his temperature suddenly rose to 106 F.; he died the following day. Death appeared to result from massive pulmonary embolism. Permission for necropsy was not granted.

Histologic Examination.—Biopsy of the jejunum showed the mucosa to be absent in some sections with blood clot protruding through the space. The clot contained acute inflammatory cells out of proportion to the red cells. The inflammation extended throughout the submucosa at the border. There was early degeneration of the muscle and connective tissue fiber.

CASE 5.—L. O., a Negro woman aged 34, had been observed in 1941, at which time she had a history of hematemesis and melena. An ulcer niche in the pyloric region was seen on roentgenographic examination. There was no history of ulcer symptoms. She was readmitted in 1944 with a history of persistent nausea and vomiting and a weight loss of 30 lbs. (13.6 Kg.). Contrast roentgenographic examination of the gastrointestinal tract showed no abnormalities. In 1945, a moderate dilatation of the duodenal bulb and the proximal half of the duodenum was observed by roentgenogram. At that time there was a recurrence of postprandial nausea and vomiting, but the patient refused surgery.

In 1948, the patient returned complaining of a recurrence of melena, accompanied with cramping, gnawing epigastric pain, and persistent postprandial nausea and vomiting. Physical examination was noncontributory. Roentgenographic examination of the gastrointestinal tract revealed a constriction of the proximal jejunum; there was some dilatation proximal to the constricted area indicating a partial occlusion of the lumen of the intestine. A diagnosis of jejunitis with ulcer or high intestinal obstruction was made. The patient again deferred surgery.

The symptoms of upper intestinal obstruction persisted, and in 1949 laparotomy was performed by Dr. John Durden. The distal duodenum and proximal jejunum were markedly dilated, and the wall of these areas was slightly thickened. Immediately distal to the dilated area was a narrowed area 2.5 cm. in diameter, and at this point an ulcer was found. Distal to the narrowed area the jejunum was normal. The upper jejunum was resected and an end-to-end jejunoduodenostomy established.

The patient's postoperative period was uncomplicated. At the end of six months she was asymptomatic and had gained weight.

Histologic Examination.—The specimen was a segment of the distal duodenum and proximal jejunum measuring 24.5 cm. in length. The proximal end measured 7.5 cm. in diameter and had a slightly thickened wall. The dilated portion ended abruptly 3.5 cm. from the proximal end of the specimen, at which point there was a puckered stricture with the diameter narrowed to 2.5 cm. The wall here was slightly thickened and indurated. There was a shallow ulceration with a pinkish-tan granular base and sloping edges. In the adjacent mesenteric fat 16 lymph nodes varying from 2 to 15 mm. in diameter were identified. There was evidence of minimal regenerative activity of the mucosa at the edge of the ulcer, as evidenced by a single layer of flattened epithelial cells extending over the granulation tissue for a short distance from the edge of the mucosa.

Summary of Five Cases of Nonspecific Ulcer of the Small Intestine

Case	Age, Yr.	Sex	Race	Preoperative Diagnosis	Duration of Symptoms	Location of Ulcer	Complications	Operative Treatment	Result
1	39	M	Negro	Ruptured appendix with intestinal obstruction	12 days	Ileum	Perforation	Resection, end-to-end anastomosis	Recovery
2	48	M	Negro	Mesenteric thrombosis or diverticulitis of sigmoid	1 day	Jejunum	.....	Resection, end-to-end anastomosis	Recovery
3	19	M	White	Inflammation appendiceal stump or intestinal obstruction	6 hr.	Ileum	Perforation	Resection, end-to-end anastomosis	Recovery
4	80	M	White	Strangulated hernia	5 days	Jejunum	Perforation	Transverse closure	Died after 5 days
5	34	F	Negro	Jejunitis with ulcer (X-ray) or intestinal obstruction	8 yr.	Jejunum	.....	Resection, jejunoduodenostomy	Recovery

COMMENT

The diagnosis of nonspecific ulcer of the small intestine is very difficult. A case reported by Ebeling [16] in 1933 was the first in which diagnosis was made preoperatively. In the chronic type the lesions form slowly, and months or years of vague and bizarre, but definite, abdominal discomfort, and occasionally even hemorrhage into the intestinal tract, may be suffered by the patient. On the other hand, perforation of the intestine may actually occur without previous indication of pathologic change, the first symptoms being those of perforation of a hollow viscus into the peritoneal cavity. These symptoms may simulate various acute abdominal conditions, such as ruptured peptic or duodenal ulcer or acute appendicitis.

In four of the five cases presented here the condition was acute, with perforation in three. The duration of symptoms ranged from 12 days to six hours. The chief symptoms were nausea, vomiting, and nonlocalized, persistent abdominal pain which had started suddenly and was occasionally colicky. Neither melena nor hematemesis was present. The patients had fever on admission, usually leukocytosis, abdominal distention with tenderness, rebound tenderness, and rectal tenderness. Roentgenographic studies of the abdomen were without diagnostic significance.

In the chronic case there was a history of melena and considerable weight loss. The patient was observed over

a period of approximately nine years. In the eighth year the roentgenographic studies of the gastrointestinal tract indicated a contracted area of jejunum with dilatation behind that area. A tentative diagnosis of jejunal ulcer was made. This was the only instance in the five cases reported here in which the correct diagnosis was made preoperatively. It is obvious that the symptoms in the acute cases were of little value for a specific diagnosis. Even those in the chronic case made diagnosis problematic.

As reported by others, the majority of these cases occurred in adult males.

The mortality rate from this lesion is high. Morrin [1b] reported a collection of 85 cases with 56 fatalities, or a mortality rate of 66 per cent. In the 45 cases collected by Evert and his associates [1a] following Morrin's report, there were 21 deaths, a mortality rate of 47 per cent.

Surgery is the only means by which this lesion has been successfully treated, but there is no single procedure used for its successful removal. Although the reports in the literature indicate that simple closure may reduce the risk of operation, it was the general opinion that resection would be the better procedure.

Free perforation was present in 26 of the 45 cases collected by Evert and associates. Simple closure was used in nine, with one fatality; excision of the ulcer and transverse closure in three, with one fatality; and resection and anastomosis in five, with three fatalities. In Morrin's collection there were 38 cases with perforation in which some type of operative procedure was used: suture alone in 23, with 10 fatalities; and resection in six, with two fatalities. Robinson and Wise,[2] reviewing 39 cases in 1940, found that 18 were treated by resection and anastomosis, with four fatalities, and 15 were treated by simple transverse closure, with six fatalities.

Resection and establishment of an end-to-end anastomosis was carried out in four of the five cases reported here; in two of these, perforation had occurred. There was perforation in one (case 4) in which transverse closure was used. This was the only case which terminated in death, although death appeared to result from pulmonary embolism. These five cases do not warrant definite conclusions, but the impression from them is that resection would be the procedure of choice even in patients who were poor risks for any surgery.

The patients whose cases are discussed here were prepared for operation by the routine used in this hospital for intestinal surgery, whether emergency or elective. Levin intubation with Wangensteen suction was performed, and blood and fluids were administered intravenously as indicated, depending upon the state of dehydration. The preferred anesthesia was spinal with thiopental analgesia, but gas-oxygen-ether closed inhalation was used if spinal anesthesia was contraindicated.

The patient usually is in no condition to withstand an extensive or prolonged search at laparotomy for the pathologic area, yet it is essential that the small intestine be carefully examined for these lesions when no other pathology which would account for the symptoms is found. If the possible presence of this lesion is kept in mind, the search for the affected part may be more successful.

SUMMARY

Five cases of nonspecific ulcer of the small intestine are presented. Four of the cases were acute and diagnosis was not made until laparotomy was performed. One of the cases was chronic, and diagnosis by roentgenography was made preoperatively. Resection and establishment of an end-to-end anastomosis was carried out in four cases, in all of which the patients survived. One case, in which transverse closure was performed, terminated in death.

478 Peachtree St., N.E. (3).

2. Robinson, D. R., and Wise, W. D.: Simple Nonspecific Ulcer of the Jejuno-Ileum, Surg., Gynec. & Obst. **70**: 1097, 1940.

IMMUNITY IN TUBERCULOSIS

J. Arthur Myers, M.D., Minneapolis

Dependable immunity is established against smallpox by vaccination—why not against tuberculosis? This is the question Koch asked in 1882, and it is still being asked in 1950. Literally scores of physicians and scientists have diligently sought the answer. No pathogenic micro-organism has deceived the medical profession with respect to immunity as often, as continuously or as completely as the tubercle bacillus.

HISTORICAL AND EXPERIMENTAL BACKGROUND

In 1920 and 1921 I was assigned to projects that offered unlimited opportunities to study tuberculosis in all of its stages of development. One of them was concerned with aiding in the organization and direction of a tuberculosis control program among students of the University of Minnesota; the other, with a tuberculosis control program for all school children of Minneapolis. I also participated in other projects that included persons beyond university age and even through the period of senility.

The literature revealed numerous attempts to immunize artificially against this disease. So-called immunizing agents had been produced from bacilli killed by various methods such as heat, light rays and lipid solvents. Some workers reported apparent success, but ultimately all failed.

The attempt to vaccinate with living virulent and attenuated tubercle bacilli was begun by Cavagnis in 1886. Before the nineteenth century closed, several others were active in this field. In 1902 von Behring administered intravenously to calves living human type tubercle bacilli with greatly reduced virulence for guinea pigs. Inoculation with this bovovaccine appeared to re-

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sult in definite resistance to virulent tubercle bacilli. It was recommended for and used in large numbers of cattle in several nations, and its use was made obligatory in some European provinces. Von Behring's claims were so convincing that the German government constructed a large institute where he made the vaccine for worldwide distribution. The passage of time proved that the apparent resistance produced by this vaccine was of short duration and not sufficiently effective to be of practical value. Numerous investigators entered the field, including Koch, Neufeld, Miessner, Heymans, Klimmer, Arloing and Theobald Smith, seeking to modify von Behring's method so as to make it effective.

Calmette and Guérin, working with virulent bovine bacilli which Nocard had isolated from the udder of a tuberculous cow (1902), observed that the bacilli grew perfectly on a special medium. Moreover, their virulence diminished progressively until large doses did not seem to harm calves for at least 18 months. These calves also manifested apparent resistance to virulent tubercle bacilli. In 1921 Calmette named this organism BCG (bacillus Calmette-Guérin) and in 1924 declared it a *virus fixé*.

When our work began, in 1920, all the previous experiments aimed at production of dependable artificial immunity had failed. Although sweeping claims were being made for the widely discussed efficaciousness of BCG, its use was still limited to animals. We decided to support a fundamental program in which tuberculosis is treated as a contagious disease and by which tubercle bacilli are prevented from invading living human and animal tissues. Our predecessors and colleagues had completed a sanatorium building program in 1918, but there were still many persons with contagious tuberculosis unaccommodated in sanatoriums. Veterinarians were attacking the bovine type of tubercle bacillus, but there was still much contagious tuberculosis among cattle. Sanatorium staffs, public health workers, physicians in practice, nurses, social workers, veterinarians and a large segment of the citizenry of the state engaged in this fundamental tuberculosis control program.

EARLY CLINICAL TRIALS

By 1922 Weill-Halle of France was administering BCG to infants. Over the next few years the claims of Calmette and his disciples were so sweeping that we were faced with heavy pressure to infect those for whom we were responsible with this living tubercle bacillus in the hope of immunizing them. The opportunity to confer personally with Calmette as well as to hear him read a manuscript on vaccination of newborn infants came in 1926. He stoutly maintained that vaccination with BCG was harmless, that the virulence of the organisms was absolutely and permanently fixed and that efficaciousness in safeguarding infants from immediate environmental contagion had been "demonstrated beyond doubt." In our opinion Calmette had added nothing new, and his claims with reference to harmlessness and efficaciousness were not unlike those of many others who had preceded him but were later proved to be wrong.

As our tuberculosis control projects by fundamental methods progressed, we kept in close touch with reports of those attempting to immunize with BCG and later with vole tubercle bacillus. Calmette believed his vaccine to be so efficacious that when leading clinicians of France urged a carefully controlled study he refused them and encouraged mass vaccination without the benefit of scientific proof. Thus, scientists and outstanding physicians lost confidence in him. His reports of 1927 and 1928 were severely criticized by statisticians such as Greenwood and others, including the editor of the *British Medical Journal*. About the same time, Petroff stated that BCG had regained virulence in his laboratory. Then came the Lübeck disaster, in which 77 of 250 vaccinated infants died of tuberculosis (presumably from contamination of the vaccine).

Calmette's original work was on animals, particularly cattle (1908 to 1922), and he reported such good results that enthusiasm ran high. In the United States, veterinarians and others put BCG to the most rigid and carefully controlled tests, but they found it of no practical value. In fact, when subjected to the same exposure, cattle that had received BCG contracted as much destructive tuberculosis as the controls. The veterinarians of North America then proceeded with their long since established fundamental tuberculosis control measures. By such means they accomplished results that have never been attained anywhere in the world through the use of BCG.

After these catastrophes and other setbacks, enthusiasm for BCG reached a low ebb throughout the world except in Scandinavia, where Heimbeck (1927) and, later, others, including Holm and Wallgren, maintained interest. In the United States, Park and associates, of the New York City Department of Health, instituted a study on BCG among children in 1926. Rosenthal of Chicago brought BCG from France in 1934 and is now its most enthusiastic proponent in this country.

ACCUMULATION OF CLINICAL DATA

Soon after 1920 we observed that when children first became reactors to tuberculin a small percentage of them had fever, erythema nodosum, pleurisy, enlargement of hilar structures and small infiltrates in the lungs. When these manifestations subsided, the children appeared well. An occasional child had fatal pneumonia, meningitis or miliary disease. As the years passed, we observed many persons, particularly students of nursing and medicine, who first became infected with tubercle bacilli in adulthood. These adults responded exactly as children do when first infected, and the manifestations, such as erythema nodosum, pleurisy, enlarged hilar structures and the like, were essentially the same as those observed among children.

It is around these "allergic manifestations" that occur about the time hypersensitivity is first in evidence and for a short time thereafter that the BCG program has been developed for which so much enthusiasm has been manifested. A sizable number of workers have reported definitely less erythema nodosum, enlarged hilar shadows and other symptoms among children and young adults who have received BCG than among controls. Probably one reason for this observation is that after primary lesions were produced in the skin and regional lymph nodes with BCG, one would not expect as much evidence of such lesions in the lungs with markedly enlarged hilar lymph nodes. It would also seem likely that the allergy

produced by an organism of low virulence like BCG would be of less degree than that from virulent tubercle bacilli; hence, less erythema nodosum.

Another reason is the comparison of unlike conditions. It has been contended that far more tuberculosis develops among students of nursing and medicine who become infected while in school than among those who enter with infections established. The fact is overlooked that those who enter as tuberculin reactors already have tuberculous lesions containing tubercle bacilli. When they became infected earlier in life, some of them, and others in the group from which they were drawn, had such manifestations as erythema nodosum, pleurisy, enlargement of hilar shadows and primary infiltrates. An occasional one had acute fatal tuberculosis and therefore was eliminated from the group. On entrance to professional schools, a few had evidence of calcific deposits or diaphragmatic changes. The majority had no abnormal x-ray findings, but the tuberculin reaction attested the presence of lesions. The above allergic manifestations developed in those who first became infected in school, but after these manifestations subsided their tuberculosis status was the same as that of their tuberculin reactor classmates on admission—that is, both groups then had primary tuberculous lesions containing living tubercle bacilli.

Among the subjects of some of our earlier observations were 160 medical students who entered as tuberculin reactors, and in 3.1 per cent of whom reinfection type of lesions developed while they were in school or soon after graduation; whereas, among 145 who became reactors while in school, demonstrable lesions developed in 9.6 per cent. This might be interpreted to mean that more than three times as many students who became reactors while in school acquired clinical tuberculosis than did those who entered as reactors. Actually this is not true. In fact, of the 9.6 per cent in whom demonstrable lesions were seen, primary complexes and pleurisy accounted for 6.2 per cent, whereas only 3.4 per cent acquired chronic reinfection type of tuberculosis. Thus, only the 3.4 per cent is comparable with the 3.1 per cent of clinical lesions among the students who entered as reactors. Observations on students of nursing revealed almost identical findings. Reviewing the observations on our entire group of students of nursing and medicine, we have been unable to find any significant difference in the development of the chronic reinfection type of tuberculosis between those who entered as reactors and those who became reactors while in school.

A third reason could be a difference in the disease itself. No doubt erythema nodosum occurs more frequently in Scandinavia among persons who recently became allergic than happens in our area. Moreover, we have rarely seen the adult just infected in whom progressive pulmonary tuberculosis immediately developed, as is reported to occur often in Scandinavia.

Demonstrable lesions and conditions that are in evidence about the time allergy can be elicited and those that appear in the next year or so have constituted an extremely small part (not more than 3 per cent) of the lesions which develop in this vicinity (chart 1). This is the only group of lesions for which significant claims have been made for BCG.

UNDUE RELIANCE ON X-RAY FINDINGS

Unfortunately, in nearly all of the reports made to date on morbidity among both vaccinated and controls, the diagnoses of chest conditions have been made largely from x-ray shadows alone. Inasmuch as x-ray shadows are never pathognomonic, all diagnoses are subject to honest questioning unless the presence of tubercle bacilli has been demonstrated by culture or animal inoculation. Deaths reported by various authors as due to tuberculosis in both the vaccinated and the controls constitute a relatively small total number. Usually no statement has been made as to whether diagnoses were confirmed by necropsy and, if so, whether bacteriological evidence was required. Without the above confirmation, errors in diagnosis may have been frequent.

There is no evidence that BCG has any influence on the occurrence of lesions that often develop two or more years after the initial invasion with tubercle bacilli. So far, only Heimbeck has reported on such observations (1947 and 1948). He traced the nurses who had graduated from the Uleval Hospital, Oslo, Norway, since his studies began. This included those who had entered the school of nursing as reactors to tuberculin, those who

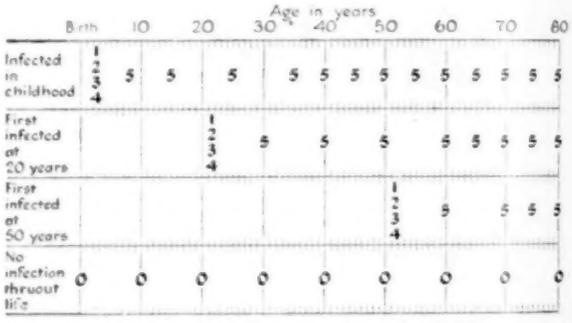

Chart 1.—Tuberculous manifestations that occur about the time a high degree of allergy is attained, regardless of age, from the first infection of the human body with tubercle bacilli. These conditions, including erythema nodosum (*1*), enlarged hilar shadows (*2*), pleurisy (*3*), meningitis, miliary disease and tuberculous pneumonia (*4*), appear promptly in only a small percentage of persons who become infected and constitute about 3 per cent of the tuberculosis problem among the groups we have observed. The reinfection forms of tuberculosis (*5*) that later occur among tuberculin reactors constitute the remaining 97 per cent of our problem. Claims that BCG will control 60 to 80 per cent of tuberculosis apply only to the 3 per cent of tuberculous manifestations that occur within a year or so after allergy can be elicited by the tuberculin reaction.

entered as nonreactors and accepted BCG and those who refused it. He found no important difference among the three groups.

Thus, claims for the efficaciousness of BCG would apply to only about 3 per cent of illness and death from tuberculosis in our area. The remaining 97 per cent have not been investigated, and evidence is lacking that this form of vaccination will be helpful in them.

NO DEPENDABLE IMMUNITY IN TUBERCULOSIS

As our work on fundamental methods of controlling tuberculosis progressed, we were unable to see any premise for attempting to produce artificial immunity because there was no satisfactory evidence that an attack of tuberculosis had ever resulted in dependable immunity. Wherever chronic tuberculosis causes death, in addition to the killing disease found at necropsy, there are always other lesions of long standing. If these lesions result in dependable immunity, why do so many die

from tuberculosis? Why is this disease of such antiquity, now the first among all diseases as a cause of incapacity and death among the people of the world as a whole? If everyone had an attack of smallpox prior to the age of 18 years, there would be practically no illness or death from this disease during the remainder of life. Yet, in areas of the world where practically everyone has had an attack of tuberculosis prior to the age of 18 years, clinical tuberculosis exists almost in epidemic form, incapacitating and killing great numbers in the remainder of life's span.

In the early days of our work we accepted the universal teaching that tuberculin reactors were immunized and advised them that they were in no danger of later having clinical tuberculosis. Many of them were apparently healthy children; but, when as young adults a considerable number returned with contagious advanced tuberculosis, we discovered that what we had taught was only theory based on speculation and personal opinions.

Between 1921 and 1941 we had under observation 18,856 children. During the first five years of this period there were 1,033 reactors who were completely examined, of whom 15 already had reinfection type of

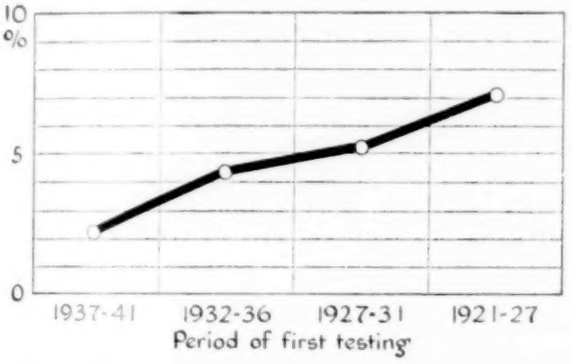

Chart 2.—Incidence of reinfection type of tuberculosis observed among tuberculin reactors. This type of disease increases with the number of years of observation after the infection is first detected.

pulmonary tuberculosis and 39 others acquired it prior to 1941. Eighteen others among the original reactors had on first examination or subsequently acquired extrathoracic reinfection type of tuberculosis.

By 1941 this study revealed that among those who reacted to tuberculin between 1921 and 1926, 7.1 per cent had at the time of the first examination or subsequently acquired the reinfection type of pulmonary or extrathoracic tuberculosis; among children first examined between 1927 and 1931, this percentage was 5.9; among those examined between 1932 and 1936 this percentage was 4.3; among those between 1937 and 1941 this percentage was 2.2. Thus, the longer tuberculin reactors remained under observation, the higher the percentage who developed clinical forms of tuberculosis (chart 2). Similar observations on other groups of people during and since this period convinced us that whatever immunity may develop in tuberculosis is not dependable or predictable.

The human body manifests its best defense against the tubercle bacilli of the first infection. When 1,000 persons (regardless of age) are infected for the first time and the tissues become hypersensitive to tuberculoprotein, a few soon have such symptoms as fever and other allergic manifestations. However, when the same 1,000 persons are reinfected exogenously or endogenously, after the tissues are allergic one may expect both acute and chronic forms of clinical tuberculosis in a considerable number within the next few decades. Acute forms of reinfection type tuberculosis, such as meningitis, miliary disease and pneumonia, may appear soon after allergy is well established. They may also emerge at any subsequent time. As the years pass, the chronic forms of disease loom. Bogen states that clinical tuberculous lesions subsequently develop in about one-half of infected persons, and that more than 10 per cent of these persons ultimately die from tuberculosis. Thus, dependable immunity is not in evidence.

NO CRITERION FOR IMMUNITY AVAILABLE

When our studies began, in 1920, several phenomena were believed to be criteria for immunity. A large amount of fibrous tissue seen on roentgenograms or at necropsy was thought to be a display of immunity. In reality, this is only a response to irritants, some of which cause more fibrosis than do the tubercle bacilli. Silicon dioxide is a good example. Deposition of lime salts was thought to be an excellent indication that immunity or resistance to tubercle bacilli had developed to a high degree. Now we know that this phenomenon is not specific for any disease. It occurs in necrotic tissue or pent-up pus from any cause. In some areas of this country more calcium deposits in the lungs and regional lymph nodes are caused by fungus infections than by tubercle bacilli.

A long interval—from months to decades—occurs between exposures to clinical tuberculosis and the appearance of illness. In many persons illness never develops. These facts were erroneously interpreted as evidence of immunity when, in reality, they indicate only that the defense mechanism has walled off the tubercle bacilli as it would silica particles. However, should the descendants of these bacilli later escape and invade allergic tissues, or should exogenous reinfection occur, there is no dependable immunity to prevent the formation of clinical lesions. For example, children who had been in intimate contact with their tuberculous mother and who appeared well long after her death were thought to have become immune; but observation revealed that during the teen ages, twenties, thirties and even later in life, enough of them fall ill from clinical tuberculosis to give the clinician no confidence in the presence of dependable immunity.

The tuberculin reaction was also regarded as a criterion of immunity. However, our observations revealed that it was only the reactors to tuberculin, children and adults alike, in whom clinical tuberculosis developed. Moreover, nearly every person in other groups of people with proved tuberculosis was allergic to tuberculoprotein. Thus it became obvious that tuberculin reactors at any age are potential patients with clinical tuberculosis. This completely shattered our confidence in the tuberculin reaction as a criterion of immunity.

Rich made an intensive study of this subject and concluded that there is no law of parallelism between hypersensitivity and acquired resistance in tuberculosis. With reference to BCG, Dubos says:

> The development of a positive tuberculin test is universally used as a criterion that the vaccine has "taken" but, unfortunately, as already pointed out, the relation of tuberculin allergy to immunity is one of the most obscure aspects of the pathogenesis of tuberculosis.

He points out that a test devised to measure streptococcal allergy does not measure resistance to streptococcus infection and by analogy the tuberculin test does not necessarily reflect immunity to tuberculosis.

Dubos says:

> . . . it is urgent that techniques be developed for the measurement of true immunity (not tuberculin allergy) in order that the BCG immunization program can be intelligently controlled. Unfortunately we are still in complete ignorance of the immunological mechanisms, humoral or cellular, which bring about immunity to tuberculosis. It is not possible, therefore, to devise any laboratory tests that would control the effectiveness of BCG vaccination.

Inasmuch as no test has been devised, we still do not know how much or how little immunity is acquired by any person. All we know with certainty is that whatever immunity develops is not dependable. Most persons who have reported results of studies on BCG and vole tubercle bacillus have observed that vaccination with these living bacilli is no more efficacious than natural infection with virulent tubercle bacilli. Since natural infection has failed to solve the tuberculosis problem, it does not seem reasonable to believe that it will be resolved by something no better. The reaction to tuberculin is only a test for allergy.

ALLERGY UNDESIRABLE

When it became obvious that allergy is not a criterion of immunity but that it is a prerequisite for the development of destructive forms of tuberculosis, we looked upon the tuberculin reaction as undesirable and therefore to be avoided if success in tuberculosis control was to be achieved. We could see no reason for deliberately producing tuberculosis with its accompanying hypersensitivity to tuberculoprotein by administering living tubercle bacilli of reduced virulence. To us this meant establishing the prerequisite for reinfection type of tuberculosis should infection with virulent tubercle bacilli occur. It was our contention that by adequate fundamental control methods we could gradually but surely prevent allergy by protecting against infection with tubercle bacilli, thus leading to the eradication of tuberculosis.

In 1925, Krause said:

> Accordingly the allergic reaction is seen to be responsible for all of the acute manifestations of tuberculosis and when so considered, it must be viewed as a potent contributor to pathogenesis. Human beings pass as perfectly well as long as they hold their tuberculous infections asymptomatic, inactive. But they are allergic, and any discharge of sufficient focal material to a new place will render them immediately ill because of the allergic reaction that ensues promptly.

This also applies to tubercle bacilli from exogenous sources.

Allergic tissues have the ability temporarily to fix or hold tubercle bacilli at or near points of lodgment. Apparently it is this fixing power which has deceived so many physicians for decades. After allergy has been artificially created and virulent tubercle bacilli are introduced, their dissemination is temporarily retarded. For example: Krause introduced tubercle bacilli subcutaneously into the groins of nonallergic guinea pigs and found that some had traveled to the lungs, tracheobronchial lymph nodes and spleen in four days or less. These animals died from tuberculosis in approximately three months. The same experiment conducted with a group of allergic guinea pigs showed that bacilli had not reached the lungs, tracheobronchial lymph nodes and spleen until three weeks to 30 days, and these animals did not die from tuberculosis until 10 to 11 months. It was the temporary fixing power of the allergy which delayed the movement of the tubercle bacilli in the allergic animals. However, the ultimate effect was the same. It is this same phenomenon which has deceived all who have attempted to produce immunity by rendering tissues allergic.

HARMLESSNESS OF BCG NOT PROVED

Although Calmette strongly maintained that BCG is harmless, we have not been able to believe that any living bacillus that was once so virulent as to cause death of animals in four to six weeks can be trusted when many millions are injected into a human body. It is now known, contrary to Calmette's belief, that the virulence of BCG changes materially. Holm says, "It is very important to keep the virulence of the BCG strain at the proper level and to watch closely the variations of its virulence." Jensen pointed out that the virulence of BCG changes and that if the strain becomes too virulent abscesses develop among those who receive it, with alarming effects. On the other hand, virulence has been found to decline so that it does not even produce allergy. Considerable effort is put forth to produce BCG of just the "right" virulence and then rush it, often by airplane, to the children who are to receive it. Not enough thought is given to how virulence of these living bacilli may change after they have been introduced into the bodies of children.

Dubos (1949) observed that three strains of BCG obtained from American collections differ significantly in their ability to produce pulmonary lesions in mice after intravenous injection. In discussing the importance of the physiological state of animals used for study, he says:

> Thus it has been shown at the Saranac Laboratory that the strain RIRV (in many respects difficult to differentiate from BCG) causes severe disease in the silicotic guinea pig but not in normal animals. It has also been found in our laboratory that one of the BCG strains commonly used for immunization can cause severe pulmonary disease (and sometimes death) when injected in sufficient amounts into mice maintained on deficient diets.

These facts we view with great alarm. Who knows what the physiological state is of the millions of children receiving BCG at this time. Concerning this, Dubos says:

> It is even conceivable that many of the classes of human beings who are most dangerously exposed to tuberculosis (for example, those living under conditions of great economic stress with inadequate food supplies) may be those least capable of responding satisfactorily to immunization procedures.

When BCG is administered subcutaneously, large abscesses at the site of injection and suppurating regional lymph nodes often occur. To avoid such extensive disease caused by BCG, Rosenthal in 1938 advocated the multiple puncture method of administration. His hope was only partially fulfilled, as such lesions still occur. For example, C. Blanch and co-workers (1948) reported 12 cases of suppurating lymph nodes complicating the administration of BCG by the puncture method. The reactions consisted of local single or multiple abscesses, infarction of lymph nodes, abscess formation and fistulization. These characteristic tuberculous abscesses

ruptured and drained for many months. Sometimes they appeared eight or more months after BCG was administered. Every administration of BCG resulting in a so-called take causes necrosis in at least the deeper layers of the skin with corresponding lesions in the regional lymph nodes.

G. Wiek (1948) reported on the various lesions found in 15 children from 5 to 14 years of age who received BCG in 1945 and 1946. Pulmonary infiltrations were seen in nine; pleurisy with effusion in four; bronchial lymph node tuberculosis in one; and elimination of bacilli without demonstrable pulmonary focus in one.

The statement is made that BCG is harmless and that no case has been reported in which it has regained virulence in man. There is a sparcity of evidence in the literature to support this statement. The number of lesions removed for biopsy or at necropsy from vaccinated persons who fell ill or died from tuberculosis to determine this point apparently is almost nil.

In animals or people in whom subsequent to inoculation with BCG progressive disease has developed, one might expect that typing of the organism would promptly reveal its identity. Unfortunately, this has not been found so simple. For example, with Dreyer's virulent BCG strains, Griffith could classify them only as mammalian and thus could not determine whether they were of human or bovine type. Holm said (1946):

> After staying in the human organism for some time the bovine type of the tubercle bacilli will change in character in such a way as to be diagnosed as the human type.

UNWARRANTED CLAIMS AND PREDICTIONS

Von Behring (1902) stated that his vaccines would soon render tuberculosis a disease of only historic interest. However, in about one decade his method was discarded.

It has been stated recently that BCG is capable of controlling 60 to 80 per cent of tuberculosis. Evidence indicates that in our area this, if proved, would apply to only about 3 per cent of our problem.

Concerning tuberculosis in Japan, it was recently said: "The BCG immunization program will in the future prevent the occurrence of most of the cases of clinical tuberculosis." Such a result has never been approached anywhere by the use of a vaccine. In Japan the annual death rate from tuberculosis is reported to have decreased from 280 per 100,000 to 181.1 in recent years. This was attributed largely to the use of BCG and would be convincing evidence were it not that similar decreases have occurred in other parts of the world in the absence of BCG. Levine says:

> We are told that in Buenos Aires the tuberculosis death rate in children under the age of 15 years decreased some 72 per cent between 1924 and 1944. This decrease was attributed to BCG. This would appear to be convincing evidence were it not for the fact that during these same years the tuberculosis death rate in New York City decreased about 95 per cent without the use of vaccine.

BCG HIGHLY CONTROVERSIAL

After more than 40 years of use of BCG in animals and more than 25 years in humans, no agreement has been reached among its proponents with reference to what constitutes a good vaccine, how it should be administered, what comprises a "take" or how long it is potent. Some physicians believe that persons should be extensively exposed to contagious tuberculous patients immediately after their tissues have been made allergic by BCG, while others contend that every effort should be made to protect against reinfection with virulent bacilli for all time after BCG has been administered. Thus, BCG lacks the qualities necessary to serve the purpose that its proponents desire. If it had been proved efficacious, its proponents should have agreed on a standard procedure; moreover, its past and present opponents would have accepted it. The medical profession has never been hesitant in accepting diagnostic, therapeutic or preventive measures of proved value. Good examples are the roentgenogram in diagnosis, insulin in therapy and smallpox vaccine in prevention. At this moment there is probably nothing in medicine as highly controversial as the value, if any, of BCG. Palmer, of the United States Public Health Service (1949), says:

> First it must be admitted in clear and unequivocal terms that strict scientific proof of the efficaciousness of BCG vaccination in the control of human tuberculosis is not available.

TOO LITTLE TO GAIN—TOO MUCH TO LOSE

Analysis of the scores of reports on the use of BCG has convinced us that any possible gain would be far too slight to justify its administration to our children, students or older adults. Tuberculosis (within a year or so after infection occurs) in which benefits are claimed from BCG constitutes only about 3 per cent of our problem. There is a total lack of evidence of any efficaciousness in the remaining 97 per cent. Even in the 3 per cent group, no one has claimed complete protection. All admit that illness and death occur among the vaccinated. Thus, even in this small group (3 per cent) one could never hope to eradicate tuberculosis by the use of BCG.

Our program of tuberculosis control would have suffered great loss had BCG been adopted. The mere finding that the skin is allergic to tuberculoprotein is proof positive of the presence of at least primary tuberculous lesions, and in most cases these lesions contain living tubercle bacilli. Therefore, the tuberculin test is our most effective weapon against tuberculosis. In diagnosis it is indispensable. It is by far the finest epidemiological agent, and it is the only accurate method of determining with reasonable promptness the effectiveness of tuberculosis eradication measures.

BCG does not prevent subsequent infections with virulent tubercle bacilli. However, it renders the tissues allergic to tuberculoprotein so that they react to tuberculin, thus depriving the individual of the inalienable right to have the presence of virulent tubercle bacilli detected promptly if and when they invade his body. Current attempts to differentiate between infections with BCG and virulent tubercle bacilli by color, degree of reaction or other means do not seem promising in the light of variations in virulence of BCG.

It is contended that only experts, individually or in teams, can satisfactorily administer BCG. This eliminates the family physicians—the general practitioners—who are so needed to solve the tuberculosis problem everywhere. In our program general practitioners have done a large volume of the work. To eliminate them by introducing a procedure that they cannot properly use and depriving them of their most potent weapon, the tuberculin test, would be disastrous.

When the physician uses the words "vaccination" and "immunity," the public thinks of almost total immunity, as obtains in smallpox. Therefore, when these words are used in association with tuberculosis, a false sense of security is transmitted to the individual and the public, which may result in relaxation of proved sanitary measures. For example, persons found to have contagious tuberculosis have refused to leave their homes because they had been told that their children could be immunized against the disease. Again, those already in institutions have insisted upon leaving because they believed the vaccine would protect their children against tubercle bacilli.

The idea is extant that vaccination with such substances as BCG will so completely control tuberculosis that the need for sanatoriums will soon vanish. This is said already to have influenced legislators and others who would ordinarily promote and support repairs and additions to existing hospitals and sanatoriums and the construction of new ones when needed.

CONTROLLED STUDY IMPERATIVE

Since Calmette opposed a controlled study in France in the early 1920's, no such investigation has been conducted with sufficient care to rule out all other factors or long enough to justify dependable conclusions regarding the efficaciousness of BCG. In fact, several proponents of its use now oppose such a study on the ground that time should not be lost while waiting for facts. This is an example of enthusiasm having outrun not only due caution but judgment as well. With our present lack of knowledge of immunity in tuberculosis, it is doubtful whether such a study is possible. Dubos, of the Rockefeller Institute for Medical Research (1949) said:

> There have been endless controversies concerning the dangers and effectiveness of immunization with the BGG vaccine. Surprisingly enough, however, little has been said or written concerning the immunological basis and the technical problems involved in the preparation of the vaccine or the measurement of its protective efficacy. Opponents as well as proponents of BCG vaccination seem to accept implicitly that the technical procedures advocated by Calmette and his associates for growing the microorganism, preparing suspensions of it and assaying the development of immunity, are fundamentally adequate and need not be modified except in detail. This is a questionable assumption and there is much reason to suspect that many of the controversies elicited by BCG vaccination cannot be settled until a number of bacteriological and immunological problems have been solved. . . . It is highly desirable, therefore, that BCG preparations be standardized in terms of their ability to induce immunity against tuberculous infection. Surprisingly enough, there is as yet no well defined technique for such an assay.

These statements from such excellent authority strongly suggest that before a dependable controlled study is possible the entire subject of immunity in tuberculosis ought to be referred to competent bacteriologists and immunologists for the determination of facts that we do not now possess. The Oxford University investigators of the vole tubercle bacillus vaccine said: "To plan a controlled experiment in tuberculosis vaccination in man is a matter of so much difficulty as to be virtually impossible." Medlar states: "The difficulties of assessing the value of vaccination with BCG in experimental animals under controlled conditions emphasizes the greater difficulties encountered in experiments in man because of the existence of uncontrolled and uncontrollable conditions."

Although the handicaps to an assessment of facts are obvious, the Division of Tuberculosis of the United States Public Health Service called a Conference on BCG in September, 1946. On recommendation of this conference, arrangements were made to set up the best controlled study possible on a limited number of persons and to employ an adequate staff in order to make sure that every person was kept under close surveillance throughout the duration of the project. It was realized that this would need to be continued for 25 years and probably longer before the desired facts about BCG would be obtained. This study is now in progress, and it deserves the unstinted support of the medical profession everywhere, since it promises the best that can be done under existing circumstances.

Much of the present enthusiasm for BCG stems from a few workers in Scandinavia. In fact, Holm, through the Danish Red Cross, has directed teams in many nations in Europe, Asia, Central America and elsewhere. The mission of these teams is not only to administer BCG to millions of children and young adults but also to convince professional workers and the public everywhere of its efficaciousness. The tuberculosis situation in Denmark is comparable in most respects to that of Minnesota. The population of the two areas is nearly equal—approximately 3,000,000. A preponderance of Minnesotans are of Scandinavian ancestry. Fundamental control measures have been similarly practiced. In neither Denmark nor Minnesota was there increase in tuberculosis mortality during World War II. The significant difference is that BCG has been used in Denmark since 1927, sparingly at first but extensively over the past decade. Thus we welcomed the opportunity to compare results in Denmark and Minnesota.

In Denmark there has been an almost continuous decrease in mortality (except during World War I) since 1906. The mortality rate in 1949 was 19 per 100,000. The mortality rates graph shows no indication of sharp decrease with the use of BCG in Denmark. Thus, a fair question is whether the fundamental methods that were operating so effectively prior to the BCG era are not actually the factors responsible for the continued decrease during this era. Holm states that at the age of 14 years, about one third of the Danish population reacts to tuberculin while two thirds are reactors at 20 to 23 years.

RESULTS FROM OUR FUNDAMENTAL METHODS ALONE

As our work progressed, the theory that primary infection immunizes was frequently emphasized and it was often said that protecting children against infection would soon lead to so many "nonimmune" adults that should they then become infected they would be swept away in vast numbers by tuberculosis. Theorists argued that producing dairy products without the bovine type of tubercle bacilli would deprive children of immunity. The theory proved to be untenable. As tuberculosis among cattle was controlled almost to the point of eradication throughout the state, there followed a decline in mortality, morbidity and infection attack rates among human beings. As the infection attack rate was further decreased by protecting children and adults alike from

human type tubercle bacilli, there also followed a corresponding decrease in morbidity and mortality from tuberculosis.

As uninfected children attained adulthood they presented no tuberculosis problem. Those, such as students of nursing and medicine, who became infected as adults, controlled primary lesions in the same manner as those who had been infected in childhood. We have not observed that primary infections in adults are more dangerous than in children or that a considerable number of the cases of tuberculosis in adults are closely related in point of time to primary infection. Thus, the theory that producing "virgin soil" in human beings would later result in disaster has proved to be a myth.

Theorists maintained that in such situations as obtain in schools of nursing and medicine and in general hospitals it was impossible to prevent exposure to tubercle bacilli. Hence the term "unavoidable exposure" was coined. When the fundamental method of control was applied, it was found practically as effective in such institutions as in the general population. In our school of medicine, in which up to 65 per cent of those who entered as nonreactors to tuberculin became reactors and 4 to 10 per cent acquired demonstrable lesions before graduation, the infection incidence was reduced to 3 per cent, and seven years have elapsed with development of a demonstrable lesion in only one student.

In a school of nursing from which no student had escaped infection and in which from 12 to 19 per cent presented demonstrable lesions before graduation, the infection attack rate was reduced to zero in the graduating class of 1945, and in only one student has a lesion been detected by roentgen shadow in the past seven years. No school of medicine or nursing has reported comparable results from BCG.

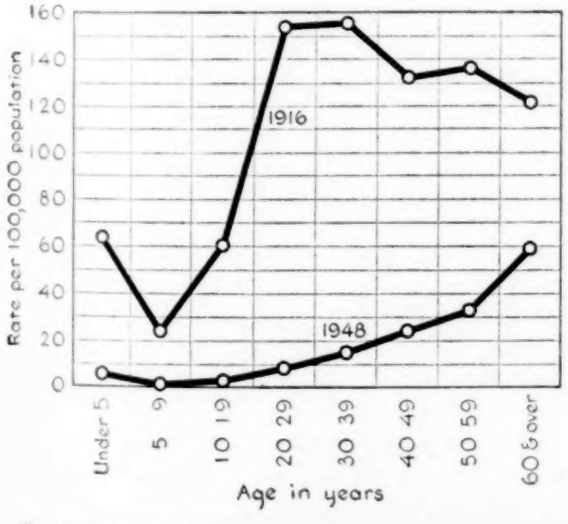

Chart 3.—Marked reduction in mortality rates among persons at various ages between 1916 and 1948. This was accomplished by fundamental methods of tuberculosis control in the absence of BCG.

American Indians have been spoken of as highly susceptible and exposure among them is "unavoidable." We have not found Minnesota Indians more susceptible than Caucasians, nor has infection been unavoidable among them. They have responded to fundamental control methods exactly as have the Caucasians. What was thought to be susceptibility was the result of neglect. When fundamental methods were instituted, the response was remarkable. For example, in 1937 there were 64 deaths (mortality rate 529.2) among our 12,500 Indians, but in 1949 only 9 (rate 71.8) died. Although BCG has been extensively used among Indians in some

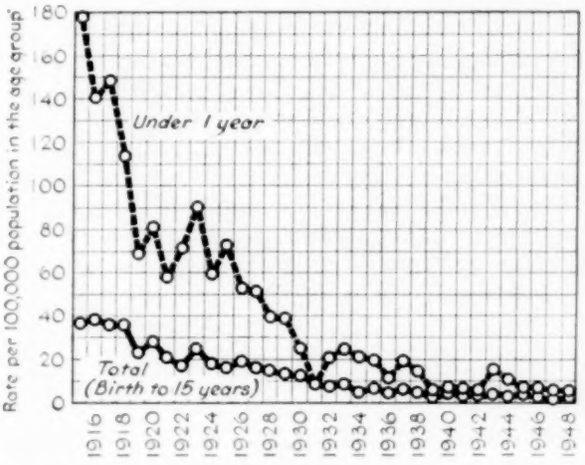

Chart 4.—Marked decrease in the tuberculosis mortality rates among children from 1916 to 1948. This was accomplished without the use of immunizing agents.

states, we know of no result comparable to that achieved in this state where it has not been employed.

Application of the same fundamental methods accomplished other excellent results. For example, by 1935 the veterinarians had tuberculosis among cattle so well controlled that the entire state was accredited. In 1949 only 0.09 per cent of the cattle reacted to tuberculin.

Waiting lists in sanatoriums decreased until many vacant beds now exist because of the lack of patients. There was a tumbling down of mortality, morbidity and infection attack rates from year to year (Chart 3). In this state the tuberculosis mortality rate dropped from 106.1 (2,400 deaths) in 1916 to 13.6 (410 deaths) in 1949. Among the 410 who died that year, 63.2 per cent were 50 years old or over and 31.5 per cent were 65 or older.

In 1916 there were 68 deaths (mortality rate 140.4) among babies under 1 year old. This was reduced to 3 deaths (rate 6.3) in 1948, and to 2 (rate 4.4) in 1949. In 1916 among children from birth to 15 years 275 (rate 38.3) died of tuberculosis, but only 19 (rate 2.6) died in 1948 and 9 (rate 1.3) died in 1949 (Chart 4). While 268 (rate 60.4) from 10 to 19 years died in 1916, only 14 (rate 2.8) died in 1948, and 7 (rate 1.4) died in 1949.

In the entire state the infection attack rate has been reduced to less than 0.3 per cent per year. By 1944 in Minneapolis only 7.7 per cent of grade school children had primary tuberculosis (tuberculous infection), against 47.3 per cent in 1926. By 1948 there were large numbers of schools in rural counties in which no child reacted to tuberculin. Among high school students in the largest city approximately 12 per cent and in other parts of the state rarely more than 8 and often less than 4 per cent now react to tuberculin. By 1945 only 6.4 per cent of students entering the University of Minnesota were infected with tubercle bacilli.

SUMMARY AND CONCLUSIONS

Fundamental methods of controlling tuberculosis consist of treating it as a contagious disease and thus preventing bacilli from invading the tissues of people and animals. This necessitates the use of the tuberculin test, x-rays, the microscope, culture mediums, animal inoculation and other procedures, as well as isolation and treatment of patients with contagious disease in hospitals and sanatoriums. Scores of physicians have long sought methods of artificially produced dependable immunity for tuberculosis. None have succeeded, probably because natural infection with virulent tubercle bacilli does not result in dependable immunity. BCG is a living bovine type bacillus of reduced virulence. In culture mediums its virulence fluctuates significantly. When introduced into human tissues, it causes tuberculous lesions which may come under control in a few months. In some cases abscesses develop and regional lymph nodes discharge pus for many months. BCG causes progressive and killing tuberculosis in silicotic animals as well as those on deficient diets. The amount of scientific work reported to prove its harmlessness in man is almost nil. BCG or vole tubercle bacillus vaccine renders the tuberculin reaction useless as a diagnostic and epidemiological agent and diminishes or even eliminates much necessary public support by creating a sense of false security in the minds of the people.

The tuberculin reaction is not a criterion of the presence of immunity. This reaction indicates only the presence of allergy, which is a prerequisite for development of clinical forms of tuberculosis. No method of identifying or measuring immunity or resistance to tubercle bacilli is available. Most observations on BCG have been limited to those conditions that occur within a year or two after allergy is established by natural infection. Here it is said to be 60 to 80 per cent efficacious. These conditions constitute only about 3 per cent of our problem. There is no evidence that it has any value in the remaining 97 per cent.

No scientific adequately controlled study of BCG has ever been conducted over a sufficient period of time to justify conclusions regarding its efficaciousness. It is doubtful whether such a study is possible in persons living in their homes. However, the serious effort now being made by the Division of Tuberculosis of the United States Public Health Service deserves unstinted support of the medical profession.

The use of fundamental methods exclusively over the last 30 years has resulted in control of tuberculosis among our students of nursing and medicine, American Indians, the general public and domestic animals that has never been remotely approached anywhere by the use of an immunizing agent. These methods when properly executed are sure of accomplishment. At the present time they afford our only hope of eradicating tuberculosis.

915 S. Seventh St.

SYPHILIS AND GONORRHEA AS CAUSES OF BLINDNESS

A STUDY OF PERSONS DECLARED LEGALLY BLIND IN OHIO

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This paper is a report of some findings concerning the role of syphilis and gonorrhea as etiological factors among persons declared legally blind in Ohio. Also considered are other aspects of syphilis and gonorrhea in blindness, including the topographical and pathological classifications of those persons whose blindness was caused by venereal disease, together with race and sex distribution of the venereal blind and observations on the age at which such persons become blind.

Financial assistance and vocational rehabilitation for blind persons in Ohio is a function of the Ohio Department of Welfare. Through the cooperation of that department and with the assistance of its Division of Research and Statistics, basic statistical data presented in this study were especially tabulated at our request and were made available to the Division of Communicable Diseases of the Ohio Department of Health. The tables presented in this study were constructed by us from the special tabulations mentioned above. We were specifically interested in bringing to light all possible information related to the role of venereal disease in blindness. The statistics of the Ohio blind are tabulated according to the standard classification of causes of blindness as recommended by the Committee on Statistics of the Blind, jointly sponsored by the American Foundation for the Blind and the National Society for the Prevention of Blindness.

When an Ohio resident applies for financial aid, he is examined by a local ophthalmologist, who reports the results of the examination on a standard form. The ophthalmologist's report is then reviewed by a consultant ophthalmologist retained by the state, and a decision is made as to whether the applicant is legally blind and entitled to financial assistance. The definition of legal blindness in Ohio is as follows: "Legal blindness is said to exist in an individual who with his better eye after correction has 20/200 vision or less, or has his field of vision reduced to a diameter of less than 20 degrees. It is also said to exist in any individual who after the removal of the lens from his eye has 20/70 vision or less."

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This definition was adopted by the Ohio Department of Welfare from certain standards suggested in the "Manual on the Use of the Standard Classification of Causes of Blindness" as published by the Committee on Statistics of the Blind.

TABLE 1.—*Etiology of Blindness in Ohio by General Classification, Jan. 1, 1939 Through Dec. 31, 1948*

General Classification	No.
Infectious diseases	852
General diseases	659
Prenatal origin	502
Trauma	326
Neoplasms	73
Poisonings	24
Undetermined or not specified	4,015
	6,442

SYPHILIS AND GONORRHEA AS CAUSES OF BLINDNESS

In the 10 year period from Jan. 1, 1939 through Dec. 31, 1948, there were 6,442 persons declared legally blind in Ohio. Etiology by general classification is shown in table 1. It will be noted that the largest group in the general classes involves cases in which the etiology was undetermined or not specified. This general group includes "unknown to science," "undetermined by physician," and "not specified." In many of these cases in Ohio, the blind person reported for his first examination many years after the onset of the condition that caused his blindness, and the examining ophthalmologist was unable to determine the etiological basis. However, this group also includes persons in whom the physician either did not take the trouble to establish a diagnosis or, if he did, failed to report his findings on the examination form. Other studies in blindness have been confronted with this same problem of having many cases of undetermined etiology.

When the 4,015 cases with undetermined etiology are deducted from the total, there are 2,427 cases of legal blindness in Ohio in which the cause was determined. Among the cases with known causes, infectious diseases

TABLE 2.—*Blindness in Ohio from Specific Infectious Diseases, Jan. 1, 1939 Through Dec. 31, 1948*

Syphilis	507 *
Trachoma	99
Ophthalmia neonatorum	68 †
Tuberculosis	50
Meningitis	23
Measles	15
Septicemia	14
Smallpox	6
Scarlet fever	5
Diphtheria	5
Gonorrhea excluding gonococcal ophthalmia neonatorum	2
Other infectious diseases, not specified	58
	852

* Of the 507 cases of syphilis, 35 cases were specified as "congenital syphilis," 31 as "acquired syphilis," and 441 as "syphilis—type not specified."

† Includes 23 cases specified as gonococcal, 40 type not specified, and 5 other types specified.

were responsible for the greatest number, namely, 852 cases. Of the total number whose blindness was caused by infectious diseases, syphilis was the etiological factor in 507 cases, which represents 59.5% of all legal blindness due to infectious diseases (table 2). Thus it is evident that among presumably preventable diseases causing blindness in Ohio, syphilis leads all others. Table 2 shows the total 852 cases of legal blindness caused by infectious diseases, with the number of cases contributed to the total by each specific disease.

When only those cases are considered whose blindness was definitely attributed to syphilis, we find that the 507 syphilitics comprise 7.9% of the total 6,442 cases, which include the 4,015 cases with undetermined or unspecified etiology. From another point of view it may be considered that the 507 cases with syphilis account for 20.9% of the total 2,427 legally blind among whom the etiology was determined. It is also reasonable to assume that a considerable percentage of the 4,015 cases whose etiology was undetermined was caused by syphilis. If the assumption is made that the distribution of etiological factors among the 2,427 cases of known etiology is a good sample of the distribution of cases among the total number of legally blind in Ohio, one could arrive at the conclusion that about one-fifth of all blindness in the state is caused by syphilis. The validity of such an

TABLE 3.—*Ten Leading Specific Causes of Legal Blindness in Ohio, Jan. 1, 1939 Through Dec. 31, 1948*

Syphilis	507 *
Prenatal origin	502
Diabetes	331
Trauma	326
Vascular disease	147
Trachoma	99
Neoplasms	73
Ophthalmia neonatorum	68 †
Tuberculosis	50
Poisonings	24
	2,127

* Of the 507 cases of syphilis, 35 cases were specified as "congenital syphilis," 31 as "acquired syphilis," and 441 as "syphilis—type not specified."

† Includes 23 cases specified as gonococcal, 40 type not specified, and 5 other types specified.

assumption has several aspects. Some ophthalmologists feel that syphilis is probably underreported. Cowan and English,[1] for example, in discussing their series of 31,352 eyes, in which the etiology was determined in 20,763 eyes, state: "We have 10,732 cases in which syphilis might have been an etiologic factor, yet it is noted as a factor in only 1,484 reports." On the other hand, it may be argued that laboratory tests for syphilis are simple, fairly accurate, and in wide use among physicians and that most syphilis among blind persons is diagnosed. Another interesting fact in the literature concerning this aspect of the role of syphilis in blindness is found in Berens' and Goldberg's [2] statement "laboratory examinations showed that Wassermann tests were made in only 2,237 (37.5%) of the 5,969 cases in which the diagnosis was an eye disease of possible syphilitic origin."

The relative importance of gonorrhea among infectious diseases causing blindness is not apparent. As may be seen in table 2, ophthalmia neonatorum as a disease entity ranked third among the infectious diseases causing blindness. Of the 68 cases ascribed to this cause, 23 were specified of gonococcal origin, and five to other specific types. The remaining 40 cases were classified as "ophthalmia neonatorum—type not specified." The preponderance of gonorrhea among those cases in which

1. Cowan, A., and English, B. C.: Causes of Blindness in Pennsylvania: Analysis of Blindness in over 30,000 Eyes, Arch. Ophth. **26**: 797 (Nov.) 1941.

2. Berens, C., and Goldberg, J. A.: Syphilis in Relation to Prevention of Blindness: A Study of 100,000 Case Records, J. A. M. A. **109**: 777 (Sept. 4) 1937.

type was specified, coupled with past medical experience and the fact that in past nomenclature the terms ophthalmia neonatorum and gonorrheal ophthalmia were practically synonymous, would lead us to believe that a substantial percentage of the 40 cases in which type was not specified was in fact gonococcal in origin. Nevertheless, when the 23 cases specified as of gonococcal origin are considered alone (which is, of course, a minimum figure), gonorrheal ophthalmia appears as an important cause of blindness due to infectious diseases. On the other hand, it should be noted that gonorrhea, excluding gonorrheal opthalmia neonatorum, was responsible for only two cases of blindness during the 10 year period.

The 10 leading causes of legal blindness (where the cause was determined and specified) are shown in table 3. Syphilis ranks first among the 10 leading specific causes of blindness in Ohio. It is striking to observe that infectious diseases, which are presumably preventable, account for four of the 10 leading causes of blindness. It may be noted also that the ophthalmia neonatorum group, which includes at least 23 cases of gonococcal origin, ranks eighth among the 10 leading causes. As an

TABLE 4.—*Number and Percentage of Cases of Legal Blindness Due to Syphilis by Year, Jan. 1, 1939 Through Dec. 31, 1948*

Year	Total No. (All Causes)	No. Caused by Syphilis	Percentage Due to Syphilis
1939	677	78	11.5
1940	726	89	12.3
1941	732	56	7.7
1942	602	33	5.5
1943	468	14	3.0
1944	380	26	6.8
1945	443	22	5.0
1946	595	56	9.4
1947	701	46	6.6
1948	1,118	87	7.8
	6,442	507	7.9

observation on "prenatal origin," which ranks second in table 3, Berens, Kerby and McKay [3] in their study on blindness among 27,002 school children indicated that syphilis is probably responsible for a large proportion of the blindness now classified as congenital or hereditary. If this latter point is true, blindness due to syphilis would loom even greater than is evidenced by the information at hand.

Table 4 shows the number and percentage of cases of legal blindness due to syphilis by year from 1939 through 1948. In interpreting table 4, one should keep in mind that the percentage of cases caused by syphilis each year is a minimum figure, since each annual total includes many cases in which the etiology was undetermined or unspecified.

Despite approximately 10 years of intensive venereal disease control activities aimed primarily at syphilis, the table shows no consistent downward trend in the percentage of blindness due to syphilis. Although there was a downward trend during the first five years of the 10 year period, the minimum percentage was reached during 1943. Considerably higher percentages have occurred in each of the five years since 1943. The percentage of blindness due to syphilis from 1939 through 1943 was 8.4. The percentage for the five years from 1944 through 1948 was 7.3. Although the most recent five year average percentage was lower, the difference was not statistically significant. On the other hand, the 7.9% of blindness due to syphilis in the 10 year period under discussion was higher than that indicated by statistics available for several years prior to 1939, during which period syphilis was responsible for 7.1% of all blindness in Ohio (table 5). The latter difference was not significant either, however.

TABLE 5.—*Legal Blindness Caused by Venereal Disease in Ohio Before 1939 and During Jan. 1, 1939 Through Dec. 31, 1948*

Cause of Legal Blindness	Before 1939 No.	Before 1939 %	1939 thru 1948 No.	1939 thru 1948 %	Both Time Periods No.	Both Time Periods %
All causes	3,137	100.0	6,442	100.0	9,579	100.0
All venereal diseases	252	8.0	532	8.3	784	8.2
Gonorrhea excluding ophthalmia neonatorum	3	0.1	2	0.03	5	0.05
Gonorrheal ophthalmia neonatorum	26	0.8	23	0.4	49	0.5
Syphilis	223	7.1	507	7.9	730	7.6

As to the role of gonorrheal ophthalmia neonatorum as a cause of blindness, the 23 cases in the 10 year period accounted for 0.4% of the total 6,442 cases. This represents a significant decrease as compared to the years before 1939 when gonorrheal ophthalmia neonatorum contributed 26 cases, or 0.8% of a total of 3,137 cases declared legally blind from all causes. There has been an even greater decrease in "ophthalmia neonatorum type not specified" since 1939. This classification (not shown on table 5 because the origin was not specified), which undoubtedly included many cases of gonococcal origin, decreased as the etiological cause of blindness from 3.1% of the total cases before 1939 to 0.6% in the 10 year period from Jan. 1, 1939 through Dec. 31, 1948.

TABLE 6.—*Comparison of Syphilis as an Etiological Factor in Blindness in Ohio and Other Areas*

Area	Total No. of Cases in Study	Cases with Etiological Factor Determined	Caused by Syphilis: No.	Caused by Syphilis: % of Total No. of Cases in Study	Caused by Syphilis: % of Cases with Etiological Factor Determined
Ohio	6,442	2,427	507	7.9	20.9
Pennsylvania [1]	31,352 *	20,763 *	1,843 *	5.9	8.9
Canada [4]	12,652	5,703	518	4.1	9.1
Massachusetts [5]	1,176	713	128	10.9	18.0
Minnesota [6]	768	563	84	10.9	14.9
Scotland (Brit. J. Ophth. 30: 381, 1946)	7,297	7,150	730	10.0	10.2
Kentucky [8]	1,901	1,037	130	6.8	12.6
Texas (Texas State J. Med. 41: 519, 1946)	6,106	1,742	411	6.7	23.6
	67,694 †	40,096 †	4,351 †	6.4 †	10.9 †

* These figures are based on the number of eyes, not the number of cases.

† Includes all cases (and eyes) in the eight studies.

It might be well to compare the Ohio figures with reported findings in other areas. Table 6 illustrates the various data concerning the percentage of all blindness due to syphilis as compared to all causes including undetermined etiology. This percentage ranges from 4.1% in the Canadian study [4] to 10.9% as determined in the

3. Berens, C.; Kerby, C. E., and McKay, E. C.: The Causes of Blindness in Children: Their Relation to Preventive Ophthalmology, J. A. M. A. 105: 1949 (Dec. 14) 1935.

4. Aylesworth, F. A.: A Study of Blindness in over 12,000 Cases in Canada, Canad. M. A. J. 54: 30 (Jan.) 1946.

Massachusetts [5] and Minnesota [6] studies. The average percentage of blindness due to syphilis as compared to all causes, including unknown etiology, was 6.4 for the eight studies combined.

AGE, SEX, AND RACE FACTORS

This section is devoted to the age, sex, and race factors of persons in our series whose blindness was caused by venereal disease. The data presented represent all cases of legal blindness in Ohio whose medical records have been coded by the Department of Welfare. They include all the cases in the 10 year period previously discussed, plus cases from 1938 and before, as shown in table 5. Coding and punching under the standard classification of Statistics of the Blind was instituted in Ohio in 1938. At that time, the backlog of cases declared legally blind during 1938 and before were coded and punched according to the standard classification, but all these pre-1939 cases were grouped together in the category of "1938 and before." The exact year was not identified. The total number of cases of legal blindness in this period before Jan. 1, 1939, was 3,137 as shown in table 5. Of this early group, 223 (7.1%) were attributed to syphilis, 26 (0.8%) to gonorrheal ophthalmia neonatorum, 97 (3.1%) to ophthalmia neonatorum type not specified, and 3 (0.1%) to gonorrhea, excluding gonorrheal ophthalmia neonatorum. Addition of these pre-1939 data to the information previously discussed shows a total of 784 legally blind persons whose disability was caused by venereal disease among all persons declared legally blind for whom records were maintained. Inasmuch as "ophthalmia neonatorum type not specified" has an unknown percentage due to the gonococcus, this group is not included in the 784 cases.

TABLE 7.—*Race and Sex Distribution of 784 Cases of Blindness Caused by Venereal Disease, Before 1938 Through Dec. 31, 1948*

	White		Negro		Other		
Venereal Disease	Male	Female	Male	Female	Male	Female	Total
Gonorrhea excluding ophthalmia neonatorum	3	1	1	0	0	0	5
Gonorrheal ophthalmia neonatorum	18	4	22	3	0	2	49
Syphilis	313	150	149	78	31	9	730
	334	155	172	81	31	11	784

Table 7 presents the sex and race breakdown of these cases for each venereal disease. We perhaps should mention here that, although the standard classification of blind statistics has a separate category for congenital and acquired syphilis, we found that relatively few cases of syphilis were so classified and that the majority were classified as "syphilis—origin not specified." We have therefore combined congenital, acquired, and type not specified, and present them as total syphilis cases.

Regardless of race, the number of males whose syphilis resulted in blindness outnumbers the females two to one. It is believed that this apparent sex difference is due to the fact that the males, being the breadwinners, are likelier to apply for financial assistance, rather than any real difference in syphilis predisposing to blindness according to sex. It should be noted here that there is a difference of opinion expressed in the literature concerning the sex distribution for all causes of blindness. Marshall and Seiler [7] reporting a series of persons applying for pensions for the blind in certain areas in Scotland stated, "It is evident there is little difference in the incidence of blindness between the sexes, and the total number of males 2,015, and females 2,063, nearly conforms to the proportion of each sex in the population." They further state, however, that the etiological factors being separately considered, acquired syphilis causes more blindness in males than females, and that blindness due to congenital syphilis, on the other hand, is commoner among females than males. (It should be noted here that in the Ohio figures in which type of syphilis was specified males predominated more than two to one in both prenatal and acquired syphilis categories.) In direct contrast to the Scotland study, Sory and Caudill [8] in their article "The Cause and Distribution of Blindness in Kentucky Among Recipients of Aid to the Needy Blind" state that "males go blind almost twice as frequently as females."

According to the 1940 census, the ratio of whites to Negroes in Ohio was 19 to 1. All other things being equal, one would expect that the ratio of whites to Negroes blinded by syphilis in this study would be the same, 19 to 1. However, as table 7 indicates, the ratio of whites to Negroes blinded by syphilis is approximately two to one for both males and females. For an estimation of the number of white and Negro syphilitics in Ohio, selective service race specific seropositivity rates were applied to the Negro and white population totals based on the 1940 census. The resulting estimates were, of course, only approximate but serve our purpose here. We were interested to observe that in the syphilis prevalence estimates by race, calculated in this manner, white syphilitics outnumbered Negro syphilitics by about two to one. This same proportion, two to one, is evidenced in table 7, which shows the number of white syphilitics blinded by their disease as compared to the number of Negroes similarly affected. From these proportions and prevalence estimates, it would appear that race is not a factor in blindness as a complication of syphilis.

Age at loss of sight due to syphilis is presented by race and sex in table 8. It occurs to us that it may be well to stress again the fact that the examining ophthalmologists in our series did not routinely specify the type of syphilitic infection causing the blindness. In most cases the type of syphilis was called "syphilis—type not determined," and only a very small percentage was specified as congenital or acquired. For this reason the entire group is shown in table 8 as total syphilis without regard for type, and the 730 represent the entire series blinded by syphilis in the period under discussion. It may be

5. Riemer, H. B. C.: Topographic and Etiologic Study of 1,176 Indigent Blind Persons in Massachusetts: Basis for Prevention of Blindness, Arch. Ophth. **32**: 304 (Oct.) 1944.

6. Stanford, C. E.: Causes of Blindness in Minnesota, Minnesota Med. **24**: 1 (Jan.) 1941.

7. Marshall, J., and Seiler, H. E.: Statistical Analysis of 3,219 Persons Certified Blind at Regional Clinic for Certification of Blind, Glasgow and South-West Scotland During 1929-1935, Brit. J. Ophth. **26**: 337 (Aug.); 386 (Sept.); 433 (Oct.) 1942.

8. Sory, R., and Caudill, F. W.: The Causes and Distribution of Blindness in Kentucky Among Recipients of Aid to the Needy Blind, Kentucky M. J. **44**: 255 (Aug.) 1946.

logical to assume that the 30 cases under 2 years of age were caused by congenital syphilis. Furthermore a majority of the 58 cases between 2 and 20 years of age were no doubt also caused by congenital syphilis. On the other hand, it seems to us that most of those cases 20 years and over were probably caused by acquired syphilis, since congenital syphilis would, as a rule, have resulted in blindness before the patients reached the age of 20. One should note that 216 of the persons were of unknown age at time of loss of sight. Once again this lack of the age at which blindness occurred points to the fact that statistics of the blind are far from perfect and that examining physicians should attempt to determine and record better histories. From a review of the table in the earlier age groups in which congenital syphilis was most probably the factor, it would appear that there are two age ranges at which blindness is likelier to be manifested, one being the category under 2 years of age and the second period beginning at about 8 years of age.

Assuming that persons 20 and over were, by and large, blinded by acquired syphilis (and excluding those persons who lost their sight before the age of 20 and the unknown group), we calculated the median age at loss of sight for each race-sex group as well as for the total. These averages were as follows: white males 42.42 years, white females 45.25 years, Negro males 41.75 years, and Negro females 36.88 years. The total white and Negro median was 42.42 years.

In order to arrive at the time lapse for an infection of syphilis to progress to the complication of blindness, we computed the median age of infection of primary and secondary syphilis reported in Ohio from the annual reports of the Ohio Department of Health. Median ages in reported infections of primary and secondary syphilis

TABLE 8.—*Syphilis as a Cause of Legal Blindness in Ohio by Race, Sex, and Age at Loss of Sight, Before 1938 Through Dec. 31, 1948*

Age at Loss of Sight, Yrs.	White		Negro		Other		
	Male	Female	Male	Female	Male	Female	Total
Under 2	10	3	11	5	1	0	30
2 to 3.9	0	2	3	0	0	0	5
4 to 5.9	2	1	3	0	0	0	6
6 to 7.9	2	3	4	0	0	0	9
8 to 9.9	5	0	6	0	0	0	11
10 to 14.9	6	0	6	1	0	0	13
15 to 19.9	8	1	3	2	0	0	14
20 to 24.9	9	4	5	3	0	1	22
25 to 29.9	12	9	8	7	0	0	36
30 to 34.9	32	10	15	11	0	0	68
35 to 39.9	23	19	9	4	2	1	58
40 to 44.9	31	11	10	8	0	0	60
45 to 49.9	19	20	9	2	3	0	53
50 to 54.9	23	18	7	4	0	0	52
55 to 59.9	19	9	10	3	1	0	42
60 and over	14	8	8	3	0	2	35
Unknown	98	32	32	25	24	5	216
	313	150	149	78	31	9	730

in Ohio were as follows: white males 28.08 years, white females 24.57 years, Negro males 24.96 years, Negro females 23.50 years, with a total for all races and sexes of 25.04 years. In comparing the median age at loss of sight of persons blinded by syphilis with the median ages of reported primary and secondary syphilis infections in the state, we established an average time period of 17.38 years between the median age of infectious syphilis cases and the median age at which the loss of sight occurred. This difference suggests that an average of 17⅓ years is required for primary and secondary syphilis to progress to syphilitic blindness among infected persons fated to have the latter complication. The 17⅓ year differential is for all races and sexes and ranges from about 13 years among Negro females to almost 21 years among white females.

TABLE 9.—*Primary Pathological Factors in 730 Cases of Syphilitic Blindness*

	No.
Optic nerve atrophy	494
Keratitis, interstitial	76
Chorioretinitis	29
Other affections of the cornea, specified	14
Iridocyclitis and uveitis	14
Choroiditis	12
Cataract	11
Hypertension (glaucoma)	9
Keratitis, unspecified	9
Retinitis	7
Optic neuritis (papillitis)	6
Keratitis ulcerative	5
Ulceration and vascularization	5
Affections of the cornea not specified	5
Iritis	5
Retinal degeneration (includes retinitis sigmentosa)	5
Disorganized eyeball (atrophic globe and phthisis bulbi)	4
Other affections of the choroid and retina, specified	3
Myopia	2
Other affections of the lens, specified	2
Affections of the choroid and retina, not specified	2
Vitreous (excluding sequellae of known ocular disease)	2
Other specified pathology	9
	730

PRIMARY PATHOLOGICAL FACTORS

In addition to the previous information concerning the 730 persons blinded by syphilis, data were also tabulated on the primary pathological factors contributing to blindness as a result of syphilis. These data are presented in table 9.

Classified nomenclature of pathological factors by site and nature of affection as recommended by the Committee on Statistics of the Blind is used in the presentation of the information. Again it is well to point out that an adequate breakdown between congenital and acquired syphilis was unavailable in our series, and for this reason the 730 cases shown in table 9 include pathological conditions resulting from both congenital and acquired syphilis.

It will be noted that optic nerve atrophy is by far the greatest primary pathological factor in blindness due to syphilis. In our series, 494 cases (67.7%), or more than two-thirds of the total 730 cases, were made blind by optic nerve atrophy. It might be noted here that this percentage of optic nerve atrophy as the pathological factor among syphilitic blind has been reported by Klauder, Meyer, and Gross [9] to be 90%. The second specific pathological cause of blindness due to syphilis is interstitial keratitis, which was responsible for 76 cases, or 10.4% of the total 730 cases. In 38 additional persons, the pathological condition was located in the cornea (including ulcerative keratitis, keratitis not specified, ulceration and vascularization, and other affections

9. Klauder, J. V.; Meyer, G. P., and Gross, B. A.: Syphilitic Primary Atrophy as a Cause of Blindness: Importance of Early Diagnosis, Am. J. Syph. Gonor. & Ven. Dis. 32: 574 (Nov.) 1948.

of the cornea specified and unspecified). This brings the total number of cases whose pathological process was located in the cornea to 114, or 15.6% of the total.

It is probably reasonable to assume that most of the persons blinded by optic nerve atrophy were suffering from acquired syphilis and that the majority of the persons blinded by interstitial keratitis were victims of congenital syphilis.

Table 10 presents the topography of pathology by general anatomic site of the 730 cases in the series. Although almost 85% of the persons were blinded by pathology located in the optic nerve, visual pathway, cortical visual center, or the cornea, it is apparent that the disease is also capable of attacking and destroying the function of all other parts of the eye. The latter point indicates again that the physicians should consider all structures of the organ of vision when examining for manifestations of syphilis in the eye.

COMMENT

In analyzing the data concerning persons declared legally blind in Ohio, we were most impressed to learn that syphilis was first among the 10 specific causes, and that one out of every five blind persons in whom the etiological factor was known was blinded by syphilis. It is to be expected that a lag would exist between improved treatment methods and the appearance of a downward trend in the incidence of syphilitic blindness. However, it must be remembered that prior to the advent of the antibiotic era, syphilis was one of the few diseases for which specific therapy was available. This fact coupled with the availability of simple and reasonably accurate laboratory diagnostic aids and a wealth of clinical experience should have brought about a decreasing incidence of syphilitic blindness. The advent of penicillin therapy and intensive venereal disease control activities during recent years should be adding impetus to this decline. Syphilitic mortality, psychoses, and cardiovascular manifestations have been reported to be declining in incidence, yet it would appear that syphilis as a cause of blindness in Ohio has not perceptibly declined.

TABLE 10.—*Topography of Pathology in 730 Persons Blinded by Syphilis by General Classification*

Anatomic Site	No.	Percentage
Optic nerve, visual pathway and cortical visual center	502	68.8
Cornea	114	15.6
Chorold and retina	59	8.1
Iris and ciliary body	21	2.9
Eyeball in general	19	2.6
Crystalline lens	13	1.8
Vitreous	2	0.3
	730	100.0

Our study suggests that an average 17 year period exists between initial infection and loss of sight, which would indicate sufficient time for the medical profession to diagnose and adequately treat these persons in order to prevent the appearance of this complication in a large percentage. This certainly presents a strong argument for intensive case-finding activity not only for the infectious stages of the disease, but also for those persons with hitherto unsuspected latent syphilis. The implied financial loss to both the person and the taxpayer who must support the afflicted is in itself another strong argument for intensification of syphilis case-finding and treatment activities by public health agencies, as well as by individual practicing physicians, in an application of the principles of preventive medicine.

Certainly there are few persons who do not consult physicians at least once in any given 17 year period of their lives. It is impossible to estimate how many of the 730 persons now blind because of syphilis would have normal vision today had their physicians maintained a high index of suspicion for the disease. Undergraduate and postgraduate medical training should be revised to include added emphasis on the diagnosis and treatment of syphilis.

Routine serologic tests assume added significance. These same 730 blind persons might have obtained an earlier diagnosis as a result of tests done for employment, hospital admission, or in accordance with premarital and prenatal laws. Premarital and prenatal examinations for syphilis have provided an effective screening process but are obviously not enough, for they are but isolated episodes in a person's life. Certainly detection of syphilis prior to the development of complications cannot be done by legislation but must be done by every practicing physician in rendering good medical care. Our study also reaffirms the findings of others that the lack of adequate recorded information seriously hampers efforts at analysis of the problem. We do feel, however, that certain valuable information is apparent.

The 7.9% of legal blindness in Ohio caused by syphilis represents a minimum figure, because many of the persons in whom cause was not specified or for whom the cause was attributed to "prenatal origin" undoubtedly were also blinded by syphilis. The figure quoted indicating that syphilis accounted for 20.9% of those persons in whom the cause of blindness was specified perhaps represents a more realistic estimate of the size of the problem.

The proportion of males to females, we believe, is of little importance as reported in the text, but the impression that race is not a predisposing factor to syphilitic blindness is valid from the evidence at hand. The preponderance of optic nerve atrophy and interstitial keratitis as pathological factors in blindness due to syphilis confirms findings reported previously in the literature.

SUMMARY

When the 6,442 persons declared legally blind during the 10 year period (Jan. 1, 1939 through Dec. 31, 1948) in whom the etiology of blindness was known were considered by general classification, infectious diseases ranked first among the general classes of causes of blindness. Among the 852 persons whose blindness was caused by infectious diseases, 507, or 59.5%, were blinded by syphilis.

Syphilis ranks first among all causes of blindness. The 507 persons blinded by syphilis represent 7.9% of all persons declared legally blind in Ohio during the 10 year period under consideration, including the large group of persons with blindness of undetermined or unspecified etiology. They, in addition, represent 20.9% of the total 2,427 legally blind among whom the etiology was determined, indicating that among those in

whom the etiological factors are known, one out of every five persons declared legally blind is blinded by syphilis.

When the percentage of persons declared legally blind because of syphilis is considered by year over the 10 year period of 1939 through 1948, no significant decrease in percentage is noted despite the fact that intensive venereal disease control activities were in effect in Ohio during the same years.

The percentage of blindness due to gonorrheal ophthalmia (0.4%) during the 10 year period under consideration is significantly lower than the percentage due to this cause (0.8%) in the several years prior to 1939.

Our study is compared in table 6 with seven other studies abstracted from the literature in order to present a composite picture of syphilis as a cause of economic blindness.

The study presents observations concerning age, sex, race, and pathological factors on 784 persons blinded by venereal disease among a total of 9,570 persons who have been declared legally blind in the state of Ohio and for whom records exist. Among persons blinded by syphilis considered in the study, males outnumber females by more than two to one. This apparent sex difference is more than likely due to the fact that the males, being the breadwinners, are more prone to apply for financial assistance rather than any difference in venereal disease predisposing to blindness according to sex. The ratio of whites to Negroes among persons blinded by syphilis considered in the study was approximately in proportion to the estimated prevalence of syphilis within each race group, which would suggest that race is not a factor in blindness as a complication of syphilis.

In a review of morbidity statistics for primary and secondary syphilis in Ohio, the median age was established at 25.04 years. Comparison of this figure with the median age at loss of sight (42.42 years) of presumably acquired syphilis cases considered in the study indicates that the infection requires an average period of time of 17.38 years to progress from the initial infection to loss of sight among those persons fated to be blinded by their disease.

The pathological factor in 67.7% of the persons blinded by syphilis was optic atrophy. Interstitial keratitis accounted for 10.4% of the total. Pathological factors in the remaining persons in the series were widely scattered as to topography and type of affection.

Throughout the study, obvious deficiencies in information were noted, indicating again that for purposes of analysis of the problem, accurate medical records must be maintained, including past medical histories and results of physical examinations, as well as accurate diagnoses.

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FATAL INJURIES IN COMPETITIVE SPORTS

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The recent deaths of two professional boxers from injuries sustained in boxing bouts stimulated a survey of similar fatalities in this sport and in other athletic competitions. The purpose of the survey was to determine the type and frequency of the injuries and to correlate them with the particular kind of athletic activity. Accordingly, these deaths may best be considered under the following classifications: (1) sports characterized by body contact—football, wrestling, boxing, hockey and basketball; (2) sports in which missiles are thrown by the hand, driven by a bat or club or shot from weapons —baseball, cricket, golf, hockey and archery;[1] (3) sports in which falls or other miscellaneous accidents may occur—horse racing, polo, handball, basketball, and track and field events; (4) aquatic competition;[2] (5) sudden and unexpected natural deaths from existing acute, subacute or chronic disease precipitated by excitement, overexertion, exhaustion, or a combination of these factors.

In New York City in the 32-yr. period from 1918 to 1950, inclusive, fatal injuries were distributed in the various sports, as seen in the table.

Fatalities

Sport	Number of Cases	Necropsies
Football	22	12
Soccer	2	2
Boxing	21	21
Wrestling	2	2
Baseball	43	27
Cricket	1	1
Golf	1	1
Basketball	7	4
Handball	3	2
Polo	1	0
Relay races	1	1
Total	104	73

From the Office of the Chief Medical Examiner.

1. Deaths in hunting accidents and in other noncompetitive activities, such as bathing, boating and fishing were not considered; nor were the sudden and unexpected deaths, unless they were associated with injuries.

2. Fatalities in hockey, aquatic competition, horse racing and archery were not observed.

SPORTS CHARACTERIZED BY BODY CONTACT

Football.—Most of the injuries in football were from direct violence in line play, in blocking and in tackling. Only on a few occasions was indirect force a factor, notably in cervical spine injuries. Other sources of direct and indirect trauma were kicks, falls, trampling, torsion and crushing force. In some cases, the manner of injury was unknown.

An acute, subacute or chronic focal infection followed by some form of generalized sepsis was a frequent cause of death in football injuries. Of ten such cases, the lower extremities were involved in five and the pelvic region in three, and there were two cases of head injuries. In this group, three instances of acute osteomyelitis were observed; one of the players developed a severe blood stream invasion and expired on the eighth day after the

injury. The primary focus demonstrated by operation was situated in the right iliac crest. In another case, a septicopyemia followed an acute osteomyelitis of the ankle, and the third developed a staphylococcic septicemia from an acute process in the right tibia. The clinical diagnoses in these cases were confirmed by operation and by necropsy.

In addition to the acute cases, there were four instances of chronic osteomyelitis that were attributed to football injuries. In one case, the necropsy revealed a low grade staphylococcic sepsis that followed a chronic suppurative and necrotizing lesion in the right iliac bone with extension to the soft parts of the iliac fossa, to the buttock and to the adjacent hip joint, where a suppurative arthritis was found. A second patient died of postoperative shock after a sequestrotomy for a chronic osteomyelitis of the femur, and a third expired of chronic sepsis also from a primary lesion in the femur. One instance of chronic osteomyelitis of the lumbar vertebras with suppurating osteoarthritis of the sacroiliac joint was encountered. This player was alleged to have received an injury to the lumbar region in a game 10 yr. prior to his death.

Both of the players in this group who had head traumas sustained injuries that resulted in infected hematomas of the scalp from which cellulitis and sepsis developed.

Intracranial injuries with hemorrhage and cerebral compression were observed on four occasions. There were three deaths from subdural hemorrhage without fracture,[3] two of which were of the acute variety in that compression symptoms developed shortly after the trauma. The other case presented the characteristics of a secondary or slow hemorrhage. The player was kicked in the head and complained of intermittent headaches but otherwise appeared in good condition. Two weeks later he began to exhibit signs of compression with headache, dizziness, occasional vomiting, hemiparesis, and stupor deepening into coma. A large subdural hemorrhage with the indications of increased cerebral pressure was found at the autopsy. In the remaining case of this series, there was a fracture of the skull with an epidural hemorrhage, which was sustained when the player ran into a wall on one side of the playing field.

Three deaths were the result of spinal injuries. There were two patients with fracture-dislocation, one involving the fourth and the other the fifth cervical segments of the spinal cord, with crushing injuries to the cord and complete motor and sensory paralysis below the level of the cord lesions, and the third had a fracture in the thoracic region, with a long-standing transverse myelitis and a urinary tract infection. The manner of injury in these cases was unknown. Such injuries are usually caused by indirect trauma from hyperextension of the neck or a twisting force applied to the head.

Intra-abdominal injuries were responsible for four deaths, and hemorrhage into the abdominal cavity was found at the necropsies on three occasions. One of these followed a rupture of the spleen from a blow on the abdomen, another presented several lacerations at the roots of the mesenteries of the small and large intestines with about 8 oz. (250 cc.) of free blood in the abdomen and an extensive retroperitoneal hematoma; the third patient had a rupture of the jejunum and the adjacent mesentery with intra-abdominal hemorrhage. The latter two were kicked in the abdomen. An acute suppurative peritonitis and septic pneumonia following a rupture of the ileum was responsible for the fourth death in this series and the patient received his injury in some unknown manner.

There was one instance of tetanus that followed a compound fracture of the right middle finger.

Injuries in soccer football were held responsible for two deaths; both players collapsed on the field. One of them had a fracture of the skull and an epidural hemorrhage and the other, alleged to have received an injury to the abdomen, showed no evidence of trauma. The necropsy established the cause of death as a luetic aortitis with stenosis of the ostia of the coronary arteries.

Boxing.—Injuries in boxing were usually the result of blows on the head or were sustained in a knockdown when the head of the boxer came in contact with the ring floor.

Of the 21 deaths in this sport, 15 were caused by subdural hemorrhage. Of these, 13 were attributed to ruptures of cortical cerebral blood vessels,[4] one to hemorrhage from a superficial cortical brain laceration, and another to a questionable injury of the posterior cerebral artery. Eight hemorrhages involved the left side and five the right, and two were bilateral. The amount of blood found in the subdural space varied from 2 to 8 oz. (60 to 250 cc.) or more. Ten of the 15 patients died within 40 hr., the period varying from one-half hour in one of the bilateral hemorrhage cases, to 38 hr. The other patient with bilateral hemorrhage survived for five and one-half hours. The remaining five patients expired after two days (in 2 cases), three days, one week and about two weeks, respectively. The latter boxer was knocked unconscious twice in a period of two weeks and apparently suffered no ill effects from the last episode. A few days later, he developed symptoms of cerebral compression and entered the hospital in an unconscious state with fixed dilated pupils, increased patellar reflexes, ankle clonus, equivocal Babinski sign, absent adbominal and cremasteric reflexes and a spasticity of the left arm. At the autopsy about 6 oz. (180 cc.) of dark red fluid blood was found under the left dura and there was marked flattening of the right cortical convolutions. Partly covering the inner surface of the dura on the left side was a brownish membrane that could be easily stripped off. Also on this side a cystlike cavity was found situated in the frontoparietal region. It measured 2½ by 4 in. (5.4 by 10.2 cm.), was covered by a delicate friable membrane and contained bloodstained fluid. The depressed base of the cyst in the frontoparietal lobes was covered by intact arachnoid membrane. In addition, the posterior border of the greater wing of the sphenoid on the left side, was rounded and thickened in comparison with the sharp-edged wing on the opposite side and the middle fossa of the skull on the left was wide and deep. A knoblike exostosis, remotely situated from the cyst and measuring ½ in. (1.3 cm.) in diameter, projected from the upper surface of the left petrous temporal bone.

3. Hey: Subdurales Hämatomals Sportverletzung, Deutsche Ztschr. ges. gerichtl. Med. **5**: 12-16, 1925.

4. Vance, B. M.: Ruptures of Surface Blood Vessels on Cerebral Hemispheres as Cause of Subdural Hemorrhage, Arch. Surg. **61**: 992-1006 (Dec.) 1950.

The position of the cystlike cavity in the subdural space and the character of the covering membrane appear to exclude porencephaly. The possibility of an old localized and encysted subdural hemorrhage was considered.

Surface injuries were noted at the autopsies of eight patients. They varied from contusions and abrasions of the forehead, eyelids, nose, lips, cheeks and ears to a ½ in. (1.3 cm.) laceration on the chin in one boxer. The remaining seven patients did not show any of these lesions. The more pronounced lacerations that induce boxing officials to terminate bouts were not observed.

Some of the patients with subdural hemorrhage exhibited other brain lesions. There were a few scattered pinhead-sized hemorrhages in the white matter of the parietal lobe in one patient, a few petechial hemorrhages in the pontile peduncle in another, very slight localized effusions of blood under the cortical arachnoid membrane in two others, and a fifth patient presented a few small hemorrhages in the lenticular nucleus. For the concepts of the production and the remote effects of these hemorrhages, the reader is referred to Leary,[5] Cassassa [6] and Martland.[7] In addition to these lesions, several patients showed streaklike hemorrhages in the pons, which were considered due to pressure effects.[8]

Ruptures of the cortical "bridging veins" were responsible for most of the subdural hemorrhages in this series.[9] Tears of the cortical arterioles have also been described by various investigators,[10] and others have advanced the theory that these ruptures were the result of anteroposterior force and oscillation of the brain,[11] or in arteries, of a sudden release of pressure after flattening of the brain against the calvarium followed by arteriolar engorgement and rupture.[12]

Various grades of hypostatic pneumonia complicated some of the delayed deaths from subdural hemorrhage.

The lesions found in the other boxers varied considerably. One heavyweight fighter suddenly collapsed in a late round of a bout and died after three days. The necropsy disclosed pinhead-sized hemorrhages in the white matter of the basal ganglions, thrombosis of a small cortical vessel situated at the junction of the temporal and occipital lobes and a wedge-shaped area of softening under the thrombosed vessel, which was interpreted as an infarct. Microscopic examination confirmed the gross findings, and, in addition, some of the small blood vessels in the white matter adjacent to the infarct showed a perivascular round-cell infiltration, and a similar infiltration was found in the meninges. Another boxer, having been knocked down, his head striking the ring floor, showed small scattered hemorrhages in the optic thalamus, the cerebellum, and the region of the fourth ventricle. He survived four hours.

Injuries of the abdominal organs are uncommon in boxers. Only one case of suppurative peritonitis from a rupture of the small intestine was encountered. The victim died six days after receiving a blow on the abdomen. Similarly, with the exception of fractures of the ribs that occur occasionally, injuries to the chest organs are very rare. They were not observed in this study.

Occasionally, the autopsy fails to demonstrate evidences of trauma in boxers who die in the ring or shortly after a bout. Such was the case in two boxers, one of whom collapsed and died in the ring, and the only lesions found were cardiac dilatation and hypertrophy. The other boxer was admitted to the hospital in an unconscious state with indefinite symptoms of cerebral compression, and the autopsy likewise failed to disclose trauma. The death was attributed to cerebral concussion. A third death from status epilepticus was also observed, in which repeated beatings in the prize ring eight years prior to the onset of the convulsive seizures, was held to be the cause.

Wrestling.—Two deaths due to wrestling were noted, one from abdominal hemorrhage and a rupture of the spleen and the other from an acute lobar pneumonia complicating a fracture of the thoracic part of the spine. In both cases, the manner of injury was unknown. The first patient survived two days and 11 hr. and the second, 11 days.

DEATHS CAUSED BY MISSILES

Baseball.—Most of the injured in baseball were amateurs or semiprofessionals. Only one major league player was fatally injured when he was struck on the head by a pitched ball. In most instances, the injury was caused by a blow from a baseball that had been thrown, or driven by a bat. A few were injured in collisions between players, in falls or when sliding into a base. Head injuries predominated, notably fractures of the skull, of which there were 25 cases. Fourteen of these were accompanied by epidural hemorrhage and two of the 14 epidural cases were complicated by direct and contrecoup lacerations of the brain with subdural and subarachnoid hemorrhage. Four others of this group showed associated brain trauma in the form of slight to moderate contusions and lacerations that were thought to be of insufficient severity to influence the diagnosis of cerebral compression from epidural hemorrhage. The characteristic localized depression in the cerebral cortex under the epidural lesion was present in all of the 14 cases.

There were 11 cases in which diagnosis was subdural or intermeningeal hemorrhage associated with fractures; seven of these were confirmed by necropsy and one by operation. Included in this group was one case of fracture with bilateral tentorial tears, subdural hemorrhage and petechial hemorrhages in the brain substance. The player with the latter condition suffered an injury to the head by a baseball and succumbed one-half hour after the accident.

Two instances of subdural hemorrhage without fracture were observed. One of these players was struck on the head by a baseball four days before entering the hospital. On admission he was unconscious and had a

5. Leary, T.: Subdural or Intradural Hemorrhages? Arch. Path. **28**: 808-820 (Dec.) 1939.

6. Cassasa, C. S. B.: Multiple Traumatic Cerebral Hemorrhages, Proc. New York Path. Soc. **24**: 101-106 (May 8) 1924.

7. Martland, H. S.: Punch Drunk, J. A. M. A. **91**: 1103-1107 (Oct. 13) 1928.

8. Wilson, G., and Winkelman, N. W.: Gross Pontile Bleeding in Traumatic and Non-Traumatic Cerebral Lesions, Arch. Neurol. & Psychiat. **15**: 455-470 (April) 1926.

9. Vance,[4] Leary.[5]

10. (*a*) Vance.[3] (*b*) Scott, M.: Spontaneous Nontraumatic Subdural Hematomas, J. A. M. A. **141**: 596-602 (Oct. 29) 1949.

11. (*a*) Trotter, W.: Chronic Subdural Hæmorrhage of Traumatic Origin, and Its Relation to Pachymeningitis Hæmorrhagica Interna, Brit. J. Surg. **2**: 271-291 (Oct.) 1914. (*b*) Grant, F. C.: Chronic Subdural Hæmatoma, J. A. M. A. **105**: 845-849 (Sept. 14) 1935. (*c*) Munro, D.: Diagnosis and Treatment of Subdural Hematomata: Report of 62 Cases, New England J. Med. **210**: 1145-1160 (May 31) 1934.

12. Werkgartner, A.: Subdural Hemorrhage from Obscure Sources, Beitr. gerichtl. Med. **5**: 191-211, 1922.

dilated right pupil and indefinite neurological signs. The necropsy disclosed more than 8 oz. (250 cc.) of fluid and clotted blood in the right subdural space, with marked flattening of the left cortical convolutions. When the brain was incised, a small amount of clotted blood was evacuated from the left temporal lobe. This lesion was interpreted as a subcortical laceration. The other player was struck by a batted ball and sustained a subdural hemorrhage, presumably from a rupture of a cortical blood vessel.

The death that occurred in a softball game was attributed to a subarachnoid hemorrhage and a fracture of the mandible. The player was injured when a bat that accidentally slipped from the hands of another player struck him on the side of the head. Aneurism of the cerebral vessels was not found.

An unusual case of traumatic hematoma of the head of the pancreas was disclosed at an autopsy. The injury was received in a collision with a player in an attempt to catch a fly ball. In addition to the hematoma, there was an extravasation of blood in the wall of the duodenum and an extensive fat necrosis. The patient entered the hospital in severe shock but survived seven days. The question of a coincidental spontaneous hemorrhage in the pancreas must be considered in such cases.

Infectious processes were responsible for 13 deaths in this game. There were six cases of osteomyelitis, five of which were acute and one chronic. The lower extremities were involved in all but one case. In the latter there were suppurative and necrotizing foci in the bodies of the ninth and 10th thoracic vertebras which followed an injury to this region by a baseball. The necropsy disclosed, besides the local lesions, thrombosis of the surrounding veins and septic infarcts of the lungs complicated by an empyema. In one of the acute cases there was a long-standing chronic osteomyelitis of the left femur, which was apparently quiescent. After an injury to the left knee by a batted ball, an acute process developed at the site of the old disease.

Acute suppurative peritonitis was the cause of death in three cases. In two of these, exploratory laparotomies revealed ruptures of the ileum. One of the players was struck on the abdomen with a bat and the other was accidentally kicked in the same region. The third player, struck by a batted ball, suffered no ill effects for five days, after which he complained of abdominal pain, with vomiting and constipation followed by meteorism and singultus. At the autopsy, a suppurative peritonitis and a perforation of the jejunum surrounded by a contused area were found. This lesion was interpreted as an original contusion, which became devitalized in the intervening five-day period and finally ruptured.

Three cases of generalized blood stream infection were attributed to baseball injuries. One was septicemia, due to a hemolytic Streptococcus following an injury to the thumb, another a staphylococcic sepsis from an infected finger and a third, a laceration of the scalp complicated by a cellulitis and a blood-stream invasion. In one instance of sudden death from a massive pulmonary embolism, thrombosis of the femoral vein and a fracture of the femur were observed, and, in another patient, a player who was struck on the chest by a batted ball and who immediately collapsed and died on the playing field, a dilated and hypertrophied heart was the only lesion found at the necropsy.

Two other deaths followed injuries by missiles. One occurred when a cricket player was struck in the abdomen by a cricket ball driven by a bat and sustained a rupture of the small intestine with acute peritonitis and the other, a caddy, died of a fractured skull and intracranial injuries when a golf ball struck him on the head.

Basketball.—Seven deaths occurred in basketball. Most of them were due to infections from relatively trivial injuries, such as a staphylococcic septicemia from an infected contusion of the thigh, a septicemia and an osteomyelitis of the femur, a similar condition with the primary lesion in the tibia, a sepsis and suppurative pericarditis following a contusion of the hip, and a post-traumatic anemia from persistent nasal hemorrhage with infection and ulceration of the nasal mucosa, which followed mild trauma to the face.

Of the two remaining patients in this category, one died of subdural hemorrhage following head trauma and the other expired from an alleged post-traumatic epilepsy supposedly due to a head injury a year prior to the fatal outcome.

Handball.—There were three deaths in this sport. Two were caused by fractures of the skull and epidural hemorrhage, one of which was subjected to necropsy and the other to operation. The third patient died of an acute leptomeningitis following a laceration of the brain without fracture. In the latter case, according to the history, the injury was sustained when a handball struck the player on the head.

A fracture of the cervical part of the spine was the cause of death of a polo player who fell from his horse, and a relay runner expired from an acute suppurative peritonitis, the consequence of a rupture of the large intestine, when he ran into railing along the side of the track.

COMMENT

The literature of the past 30 yr. contains a fair number of references to nonfatal injuries in the various branches of athletics, but fatal trauma has received scant attention, with the notable exception of the deaths in boxing. Most of the contributions in the other sports refer to isolated instances of deaths in which the circumstances are purely accidental, whereas in boxing the suspicion of a willful attempt to seriously injure an opponent may instigate a judicial inquiry, hence the medicolegal implications of such deaths have stimulated more thorough medical investigations.

A number of investigators have contributed to the study of nonfatal and fatal injuries in boxing,[13] and some of them have also considered the mechanisms of the "knockout" and their relation to the sudden deaths in the sport. Wolff,[13a] in a general review of boxing injuries, collected histories of 12 fatalities of which six were

13. (*a*) Wolff, K.: Boxsport und Boxverltezungen, Deutsche Ztschr. Chir. **208**: 379-408, 1928. (*b*) Jankovich, L.: Suites mortelles d'un combat de boxe, Ann. méd. lég. **15**: 795-799 (Oct.) 1935. (*c*) Lami, G.: Knockout e morte improuvisa nella boxe, Riforma med. **50**: 191-192 (Feb. 3) 1934. (*d*) Popielski, B.: Fatal Injuries in Boxing, Polska gaz. lek. **13**: 293-295 (April 15) 1934. (*e*) Grasso-Biondi, I.: Contributo alla conoscenza delle lesioni mortali da pugilato e dell'assurdo nella ricerca della concausa, Rassegna previd. sociale **26**: 33-60 (July-Aug.) 1939. (*f*) Tyrell, P.: Shocktod beim Boxkampf, Deutsche Ztschr. ges. gerichtl. Med. **19**: 415-421, 1932. (*g*) von Marenholtz: Tod in Boxkampf, Med. Welt. **6**: 556-557 (April 16) 1932.

caused by subdural hemorrhage, two by fractures of the skull and one each by shock, brain hemorrhage, rupture of the intestine, and edema of the glottis. The latter death, reported by von Sury,[14] followed a blow on the side of the neck, which caused hemorrhage in the larygngeal region, glottic and pulmonary edema, and death shortly after the injury was received. Lami,[13c] Jankovich,[13b] and Wolff [13a] paid particular attention to the phenomena of "knockouts" and to the causes of sudden death in boxing, while other observers discussed the lesions found at the necropsies on boxers' deaths.[13d,e]

It is generally conceded that the three kinds of trauma most likely to cause unconsciousness are blows on the chin, injury to the abdomen, and trauma to the carotid sinus. Most observers believe that sudden force applied to the chin causes cerebral concussion, reflex action on the labyrinth, and a Ménièrelike syndrome; an injury to the abdomen affects the vagus terminals and causes vasodilatation in the splanchnic area, interference with heart action and pulmonary circulation, and dyspnea. The sequence of events of trauma to the carotid sinus that was experimentally demonstrated by Hering,[15] in 1927, is regarded as a reflex ventricular fibrillation, dilatation of the blood vessels about the sinus, and a cerebral anemia. The relation of these phenomena to unexplained sudden deaths in boxers with negative necropsy findings is accepted by most investigators.

The late neurological manifestations in retired pugilists, first described by Martland [7] under the title of "Punch Drunk" and supposedly caused by repeated traumas to the head during their ring careers, stimulated the interest of a number of investigators who studied the various mental and neurological disturbances in these cases.[16] For further information on the subject, the reader is referred to their contributions.

Among those who have contributed to the subject of nonfatal injuries in football were Hanson and Hauser [17] and McPhee,[18] in their studies of the lesions among college players, and Burnett [19] and Horan,[20] who considered them among high school students. No serious craniocerebral, intra-abdominal or chest injuries were observed by these authors. Most of the injuries involved the extremities in the form of sprains, muscle and tendon ruptures, fractures, and dislocations. McPhee collected information on 889 injuries in 563 cases over a period of 10 yr. and found four fractures of the transverse processes of the lumbar vertebras, with three uneventful recoveries. In the remaining case troublesome eye symptoms developed, whose relation to the injury was questionable. The most serious injuries found in these studies were a backward dislocation of the leg at the knee with avulsion of the soft tissues and a subsequent thrombosis that necessitated amputation, two fractures of the spine, and injury to the kidney followed by hematuria. All the victims apparently recovered. Other contributors to studies of nonfatal injuries in this game were Tillman [21] and Blondal [22] and case reports of fatal traumas were published by Séjournet,[23] Frank,[24] Lande and his associates,[25] and others.

In other sports, no recorded death was found in the literature of the past 10 yr. with the exception of a case reported by Guimbellot and his co-authors [26] who reviewed the clinical observations and the autopsy findings in a case of fatal fracture of the cervical part of the spine with injury to the cord, incurred in a wrestling match. Most of the injuries were mild in nature and were troublesome mainly from the standpoint of time loss in participation in the sport.[27] On a few occasions, severe injuries with lasting effects compelled the cessation of all athletic activities.

CONCLUSIONS

It is evident that the method of play in games and the kind of athletic activity in the other competitions exerts an influence on the type of injury most likely to occur in a particular sport. Thus, subdural hemorrhage is frequent in boxing, whereas fractures of the skull and intracranial injuries predominate in baseball. Deaths from the immediate effects of trauma, such as hemorrhage and shock, and from infection were about equal in football, and infections appeared to prevail in basketball. Many of these infections followed relatively trivial injuries that would have possessed little significance had infection not set in. Since the introduction of the sulfonamides and the antibiotics, a marked reduction in the incidence of these infections has been apparent; so today they are rarely seen at the necropsies on fatal cases. Incidentally, the efforts to reduce the incidence of nonfatal accidents in the various branches of athletics by revision of the rules, by better medical supervision and coaching, and by the introduction of protective equipment have met with some measure of success.

In recent years, opponents of boxing have expressed the opinion that the sport should be abolished, that it is potentially dangerous and not necessary to the development of those attributes which are most desirable in young men. Thirty-two years of boxing competitions,

14. von Sury, K.: Boxtodesfall infolge akuten Larynxödems, Deutsche Ztschr. ges. gerichtl. Med. **1**: 695, 1922.

15. Hering, H. E.: Die Karotissinusreflexe auf Herz und Gefässe vom normal-physiologischen, pathologisch-physiologischen und klinischen Standpunkt: Gleichzeitig über die Bedeutung der Blutdrückzügler für den Normalen und Abnormen Kreislauf, Dresden, Theodor Steinkopff, 1927, pp. 141-144.

16. de Gispert Cruz, I.: Sobre la encefalopatía crónica de los boxeadores, Rev. clín. españ. **11**: 270-273 (Nov. 30) 1943. Grewel, F.: Traumatic Encephalopathy ("Punchdrunk") of Prize Fighter: Case, Nederl. tijdschr. geneesk. **85**: 154-160 (Jan. 11) 1941. La Cava, G.: La cranio-encéphalopathié traumatique des boxeurs, Bruxelles-méd. **29**: 3233-3245 (Oct. 30) 1949; 3304-3314 (Nov. 6) 1949. McAlpine, D., and Page, F.: Mid-Brain Syndrome in Professional Boxer, Proc. Roy. Soc. Med. **42**: 792-793 (Oct.) 1949. Guillain, G.; Sevileano, E., and Fandre, M.: L'encéphalite traumatique et les syndromes parkinsoniens chez les boxeurs professionnels, Bull. Acad. nat. méd. **132**: 394-406 (June 8-15) 1948.

17. Hanson, W. A., and Hauser, G. W.: Common Injuries Associated with Football at University of Minnesota, Minnesota Med. **28**: 755-758 (Sept.) 1945.

18. McPhee, H. R.: Football Injuries: 10 Year Review of Football Injuries at Princeton University, Journal Lancet **67**: 267-268 (July) 1947.

19. Burnett, J. H.: Football: Review of Injuries in Boston Secondary Schools, New England J. Med. **223**: 486-489 (Sept. 26) 1940.

20. Horan, T. N.: Analysis of Football Injuries, J. A. M. A. **103**: 325-327 (Aug. 4) 1934.

21. Tillman, G. C.: Football Injuries, J. Florida M. A. **18**: 571-574 (June) 1932.

22. Blondal, I.: Football Injuries, Ugesk. læger **105**: 932 (Sept. 9) 1943.

23. Séjournet, P.: Rupture de la rate: traumatisme minime: hémorragie lente et silencieuse; splénectomie à la 33e heure de un guérison, Bull. et mem. Soc. chirurgiens Paris **25**: 197-205 (March 17) 1933.

24. Frank, L. W.: Traumatic Rupture of Spleen: Unusual Football Injury, J. A. M. A. **116**: 1053 (March 15) 1941.

25. Lande, P.; Courbin, J., and Dervillee, P.: Mort rapide d'un joueur au cours d'une partie de football: autopsie médico-légale; rupture intrapéritonéale d'un kyste hydatique du foie, J. méd. Bordeaux **119**: 580-583 (Sept. 15-30) 1942.

26. Guimbellot, Piedelievre, R., and Derobert, L.: Fracture de la colonne cervicale au cours d'une prise de lutte, Ann. méd. lég. **21**: 117-119 (Dec.) 1941.

27. Tucker, W. E.: Cricketing Injuries, Practitioner **162**: 496-501 (June) 1949. Bennett, G. E.: Shoulder and Elbow Lesions of Professional Baseball Pitcher, J. A. M. A. **117**: 510-514 (Aug. 16) 1941.

however, have produced fewer deaths, in proportion to the number of participants, than occur in baseball or football and far fewer deaths than result from daily accidents. It seems that the moral and physical benefits derived from boxing far outweigh the dangers inherent in it or any of the other competitive sports.

125 Worth St.

CLINICAL NOTES

ASCITES DUE TO MYXEDEMA

REPORT OF A CASE

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Of the many causes of recurring ascites in patients who do not have evidence of the more common causative factors, such as cardiac, renal, or hepatic disease, myxedema is rarely considered and rarely encountered. Because of the comparative rarity of this condition the following case is reported.

REPORT OF CASE

A 45-yr.-old woman was first seen at the clinic on Nov. 1, 1949, with the chief complaints of swelling of the abdomen, weight loss, and weakness of 10 weeks' duration. She had had a thyroidectomy in 1940 for hyperthyroidism; this operation was followed by myxedema. At that time she was advised to take 1½ grains (0.10 gm.) of thyroid daily for the rest of her life. She had taken this dosage until about nine months before her present admission when an examination by her local physician revealed a basal metabolic rate of — 3. The thyroid medication was decreased to one or two ½-grain (30 mg.) tablets weekly. She had remained in fairly good health until 10 weeks before coming to the clinic, at which time total hysterectomy and bilateral salpingo-oophorectomy were performed because of a long period of menorrhagia. At the time of operation there was no evidence of malignant disease in the pelvis. About one week after the operation she noted swelling of the abdomen and considerable weakness. The abdominal swelling had increased to such an extent that it necessitated two abdominal taps in the 10-week interval; each yielded about 8 l. of yellow fluid. There was no history of abdominal pain, nausea or vomiting, and she stated that her appetite was excellent. After operation she had had three or four bowel movements daily.

The patient was admitted to the hospital on Nov. 3, 1949, for further study and observation. Physical examination on admission revealed that she was pale, the hair was coarse and dry, the skin was dry with loss of turgor, the eyebrows were sparse, and she had ascites. The heart and lungs were normal and the blood pressure was 152 mm. systolic and 100 mm. diastolic. There were no palpable lymph nodes and no palpable organs or masses in the abdomen.

Laboratory studies demonstrated a hemoglobin content of 13.5 gm., a leukocyte count of 6,700, and a normal differential count. The nonprotein nitrogen was 31 mg. per 100 cc., serum chloride 99 mEq., serum bilirubin 0.2 mg. per 100 cc., blood cholesterol 142 mg. per 100 cc., and a total serum protein of 4.9 gm., with an albumin level of 2.9 gm. and globulin level of 2 gm. Liver function tests otherwise were within normal limits. Test of the stools with guaiac gave negative results. The specific gravity of the urine was 1.018, and there was a trace of albumin; microscopic examination of the urine gave negative results. Roentgenograms of the gastrointestinal tract made after paracentesis demonstrated some forward displacement of the stomach in the region of the antrum. A roentgenogram of the chest revealed elevation of both diaphragms.

Abdominal paracentesis was performed and more than 11 l. of murky yellowish fluid having the characteristics of a transudate was obtained. Some of this fluid was injected into a guinea pig and some was centrifuged; sections made of the sediment were negative for tumor cells, as was the guinea pig injection. After the abdominal tap, examination of the abdomen revealed no palpable organs or masses.

The question then arose as to whether or not the ascites was the result of myxedema. Since the ascites had begun one week after the pelvic operation, and the patient had been taking one or two ½-grain (30 mg.) tablets of thyroid per week until two weeks before entry and was gaining 2 lb. (0.75 kg.) in weight per day, surgical exploration of the abdomen was decided upon.

Operation was performed on Nov. 17, 1949. No definite cause of ascites was found. The portal pressure was recorded as 90 mm. of water. Biopsy specimens of the liver, head of the pancreas, and enlarged nodes of the small bowel were reported as negative. The peritoneum was somewhat reddened and inflamed, but no definite evidence of tuberculosis could be seen. Specimens of the abdominal fluid were taken for smear, culture, and guinea pig injection, all of which subsequently proved negative for acid-fast organisms. With the clinical evidence of myxedema and the negative exploratory operation it was thought that the ascites was most likely the result of the poorly treated myxedema, and administration of thyroid, 2 grains (0.12 gm.) daily, was begun. In addition, the patient was given intravenous injections of 25 gm. of serum albumin daily, which raised the total protein to 6.5 gm., with an albumin content of 4.5 gm. and a globulin content of 2 gm. The ascites rapidly subsided, and her mental and physical appearance improved noticeably. She was discharged from the hospital on the 16th postoperative day with instructions to continue the daily dosage of 2 grains (0.12 gm.) of thyroid for two months and then to have a recheck examination and further evaluation of her dosage requirements. In a letter dated May 13, 1950, the patient stated that she had been taking thyroid as instructed and had not returned for check-up examination because she "had not felt so well in the past 15 years and did not want to be reminded of doctors." There had been no recurrence of ascites during this interval.

COMMENT

Ascites occurring as a direct result of myxedema has only occasionally been mentioned in medical literature. The earliest reference to its occurrence appears to be that of Kocher,[1] who, in 1883, reported two cases after total thyroidectomy. Faber,[2] in 1919, also reported the occurrence of myxedema after thyroidectomy in one case. Most of the references to the subject appear in the older literature, although one of the recent standard works on the subject noted its occurrence.[3]

The mechanism of production of ascites in these cases is not known, but it apparently involves all the serous surfaces, as cases have been reported associated with pericardial and pleural effusions either alone or in conjunction with fluid collections elsewhere in the body.[4] The problem of fluid in the various serous cavities raises two questions: 1. Has the myxedema caused myxedema heart disease and, as a result, congestive failure with ascites the symptom? 2. Is ascites caused directly by myxedema in the same manner as is subcutaneous edema, which is seen in this disease? [4g]

It is doubtful that congestive failure is part of the picture of true myxedema heart. In Zondek's original de-

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1. Kocher, T.: Ueber Kropfexstirpation und ihre Folgen, Arch. f. klin. Chir. **29**: 254-337 (Oct.) 1883.
2. Faber, K.: Kakexia strumipriva med sterk ascites som hovedsymptom, Ugesk. f. læger. **81**: 1403, 1919.
3. Means, J. H.: The Thyroid and Its Diseases, ed. 2, Philadelphia, J. B. Lippincott Company, 1948, chap. 9, pp. 211-212.

scription of myxedema heart, there was no definite evidence of congestive failure, and no mention was made of fluid in the abdominal or pleural cavities [5] in the seven cases he reported. This also appears to have been true in several cases reported by others, including Faber,[2] Mussio Fournier,[4a] Marsh, [4b] Evans,[4d] and Hatlehol.[6]

Willius and Haines,[7] in a study of 162 cases of myxedema heart, found only one patient with signs of definite congestive failure, and this patient had advanced cardiovascular renal disease. In Christian's series of 32 cases none showed evidence of congestive failure or ascites.[8] Fahr,[9] on the other hand, reported that 75% of his patients with myxedema showed some manifestations of heart failure and 30% of all his patients with myxedema had shown very severe symptoms and signs of heart failure that were promptly relieved by administration of thyroid. In support of his view he referred to the experimental work of Goldberg,[10] who removed the thyroids of sheep and goats immediately after birth and examined these cretin animals at necropsy one to two years later. Pronounced arteriosclerosis was found, in addition to generalized anasarca and stasis of liver, kidneys, and lungs. In nearly all the animals the heart was described as dilated, flabby, and pale. Hanssen,[4g] however, believed that the pronounced arteriosclerosis present in these cases after removal of the thyroid was the cause of cardiac insufficiency. He thought that the experimental observations were in accord with clinical experience that myxedema causes early and widespread arteriosclerosis that does not diminish after thyroid therapy. This was also the opinion of Christian,[11] who stated that "the myocardial disturbances caused by thyroid deficiency and cured by thyroid feeding are relatively rare, while arteriosclerosis and coronary disease coincident with thyroid deficiency are much more common."

Mussio Fournier [4a] stated that the action of thyroid extract on dropsy syndromes developing independently of thyroid insufficiency was undeniable but inconstant and that it rapidly lost its power; but, when thyroid extract was administered to a patient with a dropsy syndrome that had developed on a myxedematous basis, the action of the drug was constant and lasting. This appears to be borne out in the cases reported by Hanssen, in which ascites recurred and disappeared as thyroid was stopped or administered. When thyroid was given continuously, ascites permanently disappeared. This also appears to be true in the case here reported, as ascites did not recur after thyroid in proper dosage was begun until the patient herself decreased this dosage some seven months before she came to the clinic. It must be concluded that the true thyroid heart is a rare phenomenon and that, when present, it is rarely associated with congestive failure.

The cardiac output in cases of myxedema heart has been studied and found to be diminished [12] but without increase in the venous pressure.[13] In the case reported by Evans [4d] a series of venous pressures was determined for over a month; the pressures remained at a constant level in spite of administration of digitalis. Blumgart [14] also described normal venous pressures in seven myxedematous patients in whom there were no signs of congestive failure. Capillary filtration may be decreased [15] and the capillaries constricted.[16] The osmotic pressure of the blood [17] and the albumin content of the blood serum [18] tend to be increased. Careful observations of the type of ascitic fluid found in myxedematous ascites show nothing to distinguish it from ascites resulting from other causes.[19] A high protein content was reported in one case but not in the cases reported by others, including Faber [2] and Hanssen.[4g] It has been shown that a high protein level, as was found in this case, is not unusual in fluid of this density, namely 1.020.[20] The amount of ascitic fluid that may be found in these cases is sometimes surprising. In Hanssen's case,[4g] over an eight-year period before the diagnosis was finally established, the patient had a total of 157 paracenteses, yielding 1,357 l. of ascitic fluid, and in a case recently reported by Paddock [20] a single abdominal tap yielding 8 gal. (30 l.) of ascitic fluid.

SUMMARY

A case of ascites resulting from myxedema has been presented. It is of interest that the ascites followed a pelvic operation by one week. With the administration of thyroid, the patient has shown improvement, clinically, and the ascites has not recurred over a period of six months.

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4. (*a*) Mussio Fournier, J. C.: Syndrome hydropigène et insuffisance thyroïdienne, Bull. Acad. de méd., Par. **93**: 691-694 (June 16) 1925. (*b*) Marsh, H. E.: Myxedematous Ascites Removed by Thyroid Extract, Am. J. M. Sc. **172**: 585-588 (Oct.) 1926. (*c*) Gordon, A. H.: Some Clinical Aspects of Hypothyroidism, Canad. M. A. J. **20**: 7-10 (Jan.) 1929. (*d*) Evans, W.: Case of Myxedema with Ascites and Atony of Urinary Bladder, Endocrinology **16**: 409-416 (July-Aug.) 1932. (*e*) Freeman, E. B.: Chronic Pericardial Effusion in Myxedema: Report of Case, Ann. Int. Med. **7**: 1070-1079 (March) 1934. (*f*) Escamilla, R. T.; Lisser, H., and Shepardson, H. C.: Internal Myxoedema: Report of Case Showing Ascites, Cardiac, Intestinal and Bladder Atony, Menorrhagia, Secondary Anemia and Associated Carotinemia, ibid. **9**: 297-316 (Sept.) 1935. (*g*) Hanssen, P.: Myxoedema and Ascites, Acta med. Scandinav., supp. 90, pp. 277-287, 1938. (*h*) Marzullo, E. R., and Franco, S.: Myxedema with Multiple Serous Effusions and Cardiac Involvement (Myxedema Heart): Case Report, Am. Heart J. **17**: 368-374 (March) 1939.

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14. Blumgart, quoted by Evans, W.[7]

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COUNCIL ON PHYSICAL MEDICINE AND REHABILITATION

The Council on Physical Medicine and Rehabilitation has authorized publication of the following reports.

RALPH E. DEFOREST, M.D., *Secretary.*

HELIO-THERM, MODEL F, ACCEPTED

Manufacturer: Ultrad Laboratories, 208 Maplewood St., Watertown 72, Mass.

The Helio-Therm, Model F, is a generator of radiations including the near ultraviolet, the visible and the near infrared wavelengths. The radiation comes from an incandescent tungsten filament 300-watt lamp. The lamp (bulb) operates at a voltage higher than its rated voltage. The tungsten is "superheated" by electric current passing through the filament, giving a temperature which is stated by the firm to be approximately 3,200° Kelvin. The inside of the lamp is partly coated with aluminum to make a reflector and the emerging beam is passed through 5 cm. (2 in.) of glycerin. This filtration is intended to remove the longer infrared wavelengths. Because of the "superheating" the life of the lamp is shortened to an average of about 25 hours, according to the manufacturer. The incandescent tungsten lamp burning at a higher voltage produces an increased amount of radiation throughout the whole spectrum.

The Helio-Therm, Model F, is a floor lamp standing on a four-legged castered base. A horizontal arm, which can be raised or lowered, bears the generator. The latter is shaped like a large automobile headlight, and can be aimed in various directions. Unpacked, the device measures 162 by 65 by 72 cm. (65 by 26 by 29 in.) and weighs 13.1 kg. (29 lb.). Crated for shipment it measures 60 by 45 by 60 cm. (24 by 18 by 18 in.) and weighs 18 kg. (40 lb.). The foreign shipping weight is 40 kg. These shipping weights include the chrome tube stand (5 ft. in length, separately packed) and an extra bulb. The device will operate on either direct current or alternating current of any usual frequency, and it draws 300 watts.

In a laboratory acceptable to the Council, the spectral distribution of the energy from the lamp was examined both with and without the glycerin filter by means of a quartz spectrometer. It was found that the band spectrum extends from about 3,100 angstroms to about 14,000 angstroms without the glycerin filter and to about 11,000 with the glycerin filter.

The manufacturer submitted data showing that of the total radiant energy emitted by the Helio-Therm, Model F, in the spectrum bounded by 3,100 angstroms on the ultraviolet side and 14,000 angstroms on the infrared, approximately 2% of the total energy is in the near ultraviolet zone (3,100 to 3,900 angstroms), roughly 37% is in the visible zone (3,900 to 7,600 angstroms) and 61% in the near infrared zone (7,600 to 14,000 angstroms) which has been filtered out by the glycerin cell. The total intensity at 2 in. treatment distance is 3.2 gram calories per square centimeter per minute, or approximately three times the intensity of sunlight.

Exploratory measurements of the radiation energies were made for given distances, from which it appears that for short distances (5 cm. to 15 cm.) the radiation intensity per square centimeter from the Helio-Therm, Model F, is at least equal to that of sunlight measured under similar conditions.

While the apparatus is acceptable as a source of radiant heat for local application, the Council was not convinced that this "superheated" tungsten lamp is more efficacious therapeutically than a similar high powered (500 or 1000 watt) tungsten filament incandescent lamp operating at rated voltage, with a filter, at a treatment distance, namely, that distance between the lamp and the patient at which one approaches the threshold of pain due to excess temperature rise in the skin and blood stream.

Although, in the opinion of the Council on Physical Medicine and Rehabilitation the firm has not produced critical evidence that the radiation from a "superheated" tungsten filament lamp has superior therapeutic qualities, the Council voted to include the Helio-Therm, Model F, in its list of accepted devices as a local applicator of radiant heat.

MICROTONE HEARING AID, MODEL T-10, ACCEPTED

Manufacturer: The Microtone Company, Ford Parkway on the Mississippi, St. Paul 1.

The Microtone Hearing Aid, Model T-10, is a hearing aid of conventional design, one distinctive feature being the absence of a tone-control. The wheel-type control is for Off, On, and Gain. Excluding the latch on top and the clothing clip on the front it measures 63 by 44 by 21 mm. The transmitter alone weighs 70 gm. With the batteries it weighs 101 gm., and the receiver and cord add 12 gm.

Microtone Hearing Aid, Model T-10

From acceptable sources the Council obtained evidence that the instrument was of good construction, performed as represented by the manufacturer, and satisfied the published minimum requirements of the Council for acceptable hearing aids. The Council on Physical Medicine and Rehabilitation voted to include the Microtone Hearing Aid, Model T-10, in its list of accepted devices.

EDIN ELECTROCARDIOGRAPH, MODEL 220, ACCEPTED

Manufacturer: Edin Company, Inc., 207 Main St., Worcester 8, Mass.

The Edin Electrocardiograph, Model 220, is a direct writing instrument. The acceptance of a previous Model 210T, which likewise produces pen-and-ink records on permanent graph paper and has the same power consumption, was announced in THE JOURNAL of May 14, 1949. The present Model 220 weighs 13 kg. (29¾ lb.) and measures 25 by 21 by 43 cm. (10 by 8⅜ by 17 in.). When packed for domestic shipment it weighs 16 kg. (35 lb.) and measures 34 by 38 by 49 cm. (13½ by 11¼ by 19½ in.). The foreign shipping weight is 27 kg. (60 lb.). Included in these shipping weights are accessories.

Edin Electrocardiograph, Model 220

Evidence that the apparatus was sound in construction and satisfactory for its purpose was obtained from laboratories and clinics acceptable to the Council. The Council on Physical Medicine and Rehabilitation voted to include the Edin Electrocardiograph, Model 220, in its list of accepted devices.

MAICO HEARING AID, MODEL J, ACCEPTED

Manufacturer: The Maico Company, Inc., 21 North Third St., Minneapolis 1.

The Maico "Top Secret" Hearing Aid, Model J, is an electric hearing aid of conventional form except for the position of the microphone, which is mounted on the top of the case facing upward. The microphone grill is deeply recessed. This is intended to reduce clothing noise. The case measures 74 by 45 (greatest thickness) by 28 mm., and is somewhat wedge-shaped. The weight is 92 gm. without batteries and 123 gm. with batteries. Both magnetic and crystal receivers are available.

Maico Hearing Aid, Model J

The Council obtained evidence from acceptable sources that the workmanship and performance were good and that the instrument satisfied the published minimum requirements of the Council for acceptance of hearing aids. The Council on Physical Medicine and Rehabilitation voted to include the Maico "Top Secret" Hearing Aid, Model J, in its list of accepted devices.

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INCIDENCE OF PULMONARY CARCINOMA

Numerous surveys have indicated that the incidence of carcinoma of the lung has been increasing in recent years. There is no general agreement, however, as to whether this increase is real and absolute or merely apparent and relative, owing to such factors as better diagnosis, increasing clinical awareness of the condition, and the increase in life expectancy.

A recent report on the incidence of pulmonary carcinoma in 36,864 autopsies performed during the period 1918-1946 at the Los Angeles County Hospital shows that in the area served by this hospital the increase of the disease was real, absolute, and disproportionately greater than the increase in cancer in general.[1] In the 36,864 necropsies, a total of 6,023 malignant tumors were found which with few exceptions were the cause of death. Of these tumors, 508 were carcinomas of the lung, an incidence of 1.4% in all necropsies and 8.4% of all malignant neoplasms. During the period 1918-1922, cancer was reported in 11.2% of all autopsies, while during the period 1943-1946 it was reported in 20.7% of all autopsies. Carcinoma of the lung showed an even greater increase than cancer as a whole. No cases were reported during the period 1918-1922, but in succeeding years the incidence rose progressively. Thus, during the period 1923-1927, carcinoma of the lung was found in 0.6% of all autopsies and constituted 4.3% of all tumors found, while in 1943-1946 corresponding figures were 2.3% and 11.3%. When the incidence of lung carcinoma in consecutive groups of 500 malignant tumors was analyzed, a gradually increasing trend was apparent, with 10 lung cancers in the first 500 and 66 in the last. Again, when necropsies were divided into groups of 1,000, a gradual increase in lung carcinoma was apparent throughout the 32 years, with an acceleration in the period 1939-1943. Furthermore, the necropsy rate for lung carcinoma in relation to hospital admissions increased more than twice as much as did that for all cancers combined. In contrast with the findings in carcinoma of the lung, comparable increases were not found for carcinoma of the stomach, large intestine, or pancreas, or for intracranial tumors.

There was little change in the proportion of males in the necropsy population; hence it was concluded that the increase of lung carcinoma was not explicable on the basis of a shift in sex ratio. There was a gradual increase in the average age; however, if the increase of carcinoma of the lung were due solely to aging of the population, one would expect even greater increases in cancer of the large intestine and pancreas since the peak incidence of these diseases is at a later age than that of carcinoma of the lung.

Evidence was also presented to show that the increased incidence of lung carcinoma could not be attributed to improve recognition by the pathologist, to a rising necropsy-permit rate, or to a greater interest in obtaining permits in cases clinically diagnosed. Furthermore, there was no evidence indicating either a large-scale selective referral to the hospital of lung cancer cases or of selective immigration of persons having pulmonic diseases into Los Angeles County, while no known occupational hazards for lung cancer were unduly represented in the hospital population.

This apparently real increase in carcinoma of the lung in the Los Angeles hospital is in contrast to the very slight increase reported by Steiner in 5,515 necropsies performed between 1902 and 1941 at the University of Chicago.[2] It was felt that the findings represented a real difference in the two materials and possibly in the two communities, thus suggesting a condition existing in the entire population of the county. It was felt that the smog condition was of too recent occurrence to account for the increase, unless previous data on the latent period for induction of lung cancer are inapplicable. In concluding their report, the authors emphasize that, regardless of whether the incidence of lung carcinoma is increasing or has increased at all, its present level is so high that it constitutes a major public health problem requiring appropriate clinical, experimental, and statistical studies.

BACILLARY DYSENTERY

Bacillary dysentery is a disease that has been widely distributed throughout the world from time immemorial. It was known to the ancients, as shown in Hippocrates' "Epidemeion." Until the discovery of the specific causative agent by Shiga in 1898 and Flexner in 1900, very little advance was made in our knowledge of the disease. The greatest progress has been made during the last two decades, particularly in the fields of pathology and bacteriology. For example, patients may have lesions of the intestine at sigmoidoscopy with little or no clinical symptoms and signs. Yet these patients are just as infective as those who are sick. No longer is pathogenicity determined by the particular strain present, of which there are at least 19 different types. The Sonne-Duval bacillus, once regarded as causing a rather benign dysentery in children, may be as virulent as the time-honored Shiga-Kruse infection. Any dysentery strain may play havoc in a population hitherto free of the disease, and in endemic areas susceptibility appears to return about every 20 years.

The disease has been traced from the acute to the chronic phase in the same human being, so that the chronic type is now better understood. While the etiology

1. Steiner, P. E.; Butt, E. M., and Edmondson, H. A.: Pulmonary Carcinoma Revealed at Necropsy, with Reference to Increasing Incidence in the Los Angeles County Hospital, J. Nat. Cancer Inst. **11**: 497, 1950.
2. Steiner, P. E.: Incidence of Primary Carcinoma of the Lung with Special Reference to Its Increase, Arch. Path. **37**: 185, 1944

of chronic distal ileitis or chronic ulcerative colitis is still controversial, there appears to be no doubt that cases have followed acute bacillary dysentery. In the Jersey City epidemic of Flexner dysentery in 1934, chronic distal ileitis or chronic ulcerative colitis developed in 10.7%. Whether all cases follow bacillary dysentery cannot be proved, since many of the patients are seen for the first time one or more years after onset, when the dysentery organisms are no longer present and the original acute attack has been forgotten.

Bacillary dysentery continues to be a great "war disease"; it is transmitted by food, fingers, and flies and remains a serious hazard for civilians despite the sulfonamides and antibiotics.

THE CONTROL OF MALARIA

Within the lifetime of many physicians now in practice a tremendous change in the incidence of and mortality from malaria has occurred, not only in the United States but also in many other countries. Both the cause of the disease, first described by Laveran, the French Army surgeon in 1880, and its insect vectors, the Anopheles group of mosquitoes, incriminated by Ronald Ross and the Italian malariologists 10 years later, have been known for well over 50 years. With these discoveries as a basis for preventive action, it was obvious that this must take the form of eradication of the vector or sterilization of the blood of human carriers, but preferably both.

As Faust [1] has shown, there is no reasonable doubt that Anopheles mosquitoes existed in America long before the Columbian Era and that the Plasmodium was introduced by infected Europeans and African slaves. The spread of the disease from original foci in the eastern United States was mainly the result of the migrations of the pioneers, and it was known by 1850 to exist from New England and the southeastern states to California and the Columbia River Valley. In certain areas—northernmost New England, the Allegheny highlands, the Rocky Mountains, the Great Inland Desert, and the Western Sierras—it was not known to occur. Naturally it flourished best in this country in the southern states, where the Anopheles was able to breed for eight to 10 months of the year, as contrasted with the three or four warm summer months of the northern area.

A complicating factor in the distribution and survival of the disease was war, for in the conflict in the states in 1861-1865, northern soldiers returning from service in the southern states carried the infection; in the Spanish-American War many soldiers serving in Cuba and the Philippines were infected; and in World War II many northern troops were trained in the South and much of the fighting occurred in districts in the Pacific more or less heavily infected with malaria. Returning soldiers were frequently carriers, despite the prophylactic and curative use of antimalarial drugs.

The control of malaria is a good example of the fact that such problems are not merely medical but also economic. The eradication of Anopheles mosquitoes requires large sums of money not only for drainage projects but also for the purchase and proper distribution of insecticides and larvicides. This is an annual performance and needs trained personnel for adequate accomplishment. In periods of financial depression there is diminished expenditure for mosquito control and inability of the poorer segments of the population to procure properly balanced diets, with resulting lessened resistance to infection. Economic factors were doubtless responsible for the fact that widespread attempts to control malaria were not made until many years after the knowledge necessary to formulate prophylactic measures was available.

Before the war in Korea, the disease was largely under control, but one must not overlook the fact that many malaria-carrying soldiers will return from this area and that there must be constant watchfulness to see that this does not result in a flare-up in the incidence of the disease.

BCG VACCINATION

BCG vaccination has been used for more than a quarter of a century. Its true role in the prevention of tuberculosis still remains a subject of controversy. A status report has been published in THE JOURNAL [1] by the Council on Pharmacy and Chemistry. Though it is impossible to conduct ideal experimental BCG studies in man, such studies can provide evidence approaching that sought in animal experimentation. Such evidence is reported by Dahlström and Difs,[2] who investigated the incidence of various types of tuberculous disease among tuberculin-negative Swedish Army conscripts, whom they divided into two comparable groups, one of which was vaccinated with BCG. The results indicate that the incidence of not only primary tuberculosis but also exudative tuberculous pleurisy and early postprimary destructive pulmonary tuberculosis was significantly reduced in the vaccinated (36,235) compared with the nonvaccinated (25,239) soldiers. This is the first time it has been shown that the postprimary tuberculous diseases may be prevented by BCG vaccination. The number of deaths from tuberculosis was also significantly reduced. Since the vast majority of soldiers were infected while in military service, the Swedish investigators conclude that it "is therefore important to carry out vaccination, so far as possible, in such good time that protection will have developed before the conscript enters military service."

BCG vaccination seems to be gaining favor in some quarters for the prophylaxis of tuberculosis, but the disease should not be regarded as being in the class of typhoid; so far as control is concerned, much remains to be done. Other careful studies of tuberculosis prophylaxis with BCG vaccination should be instituted with the ever essential tuberculin testing and sane interpretation.

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1. Faust, E. C.: History of Malaria in the United States, Am. Scientist **39**: 121, 1951.

1. The Status of BCG Vaccine, Report of the Council on Pharmacy and Chemistry, J. A. M. A. **144**: 1260-1261 (Dec. 9) 1950.

2. Dahlström, G., and Difs, H.: The Efficacy of BCG Vaccination: A Study on Vaccinated and Tuberculin Negative Non-Vaccinated Conscripts, Acta tuberc. Scandinav. **25** (Supp. 27): 1-133, 1951.

MEDICAL NEWS

COLORADO

Annual Cancer Seminar.—This seminar will be held at the Broadmoor Hotel, Colorado Springs, Sept. 8, under the sponsorship of the Penrose Cancer Hospital and the College of American Pathologists. Dr. Arthur P. Stout, professor of pathology at Columbia University, New York, and Dr. Philip J. Hodes, professor of radiology at the University of Pennsylvania, Philadelphia, will jointly conduct the seminar. Dr. Leo H. Garland, clinical professor of radiology, Stanford University School of Medicine, San Francisco, will speak at the banquet. Fifteen problem cases of thoracic lesions having some relation to the field of tumors have been chosen. A photographic reproduction of the roentgenograms and a clinical summary of each case will be submitted to the participating radiologists, who will be invited to submit their diagnoses on an unsigned card, for purposes of tabulation. A set of slides of the same cases, with the same clinical summaries, will be submitted to the participating pathologists. All those interested may attend. There will be no registration fee; hotel reservations should be requested in advance. The meeting will be called at 9 a. m.

CONNECTICUT

Health Exhibits at County Fairs.—The Connecticut State Medical Society's committee on rural medical service will sponsor a health exhibit for display at a number of Connecticut's fairs in August and September. The project was inaugurated last year when a three-panel exhibit presented information on physician distribution in rural areas and voluntary health insurance. Success of that project has encouraged its continuance. This year's exhibit will feature a display of health pamphlets for distribution to fair patrons. Members of the committee are Drs. Edward H. Basden, Willimantic; Gaert S. Gudernatsch, Sharon; Enos J. O'Connell, Unionville; William H. Pomeroy, Poquonock, and William H. Upson, Suffield.

GEORGIA

Personal.—Dr. William L. Champion, Atlanta, said to be the oldest urologist in Georgia, on July 7 was honor guest at a dinner given by an associate, Dr. Major F. Fowler, Atlanta, at the East Lake Country Club. All members of Fulton County Medical Society were invited to the dinner, which marked Dr. Champion's 83rd birthday.

Hospital Dedicated.—The new 112-bed Hall County Hospital at Gainesville was dedicated by Gov. Herman Talmadge on Aug. 1. Heading the medical staff will be Dr. Cleveland D. Whelchel, Gainesville, with Drs. Clarence G. Butler, Walter R. Garner, Herbert E. Valentine Jr., Bradley B. Davis, and Clyde W. Whitworth, all of Gainesville, composing the executive committee. Drs. Davis and Whitworth are vice-chief of staff and secretary-treasurer, respectively.

ILLINOIS

Narcotic Violation.—Dr. Claude E. Gilliatt, Allendale, pleaded *nolo contendere* to a violation of the federal narcotic law in the United States District Court at Danville, and on June 11 he was fined $150 and costs.

University to Operate Neuropsychiatric Institute.—A bill transferring the Illinois Neuropsychiatric Institute in Chicago to the University of Illinois from the former joint operation by the university and the State Department of Public Welfare has been passed by the Illinois General Assembly and signed by Governor Stevenson. It includes an appropriation of $1,405,700 to the University of Illinois for the operation and maintenance of the institute for the biennium beginning July 1, 1951. Professional services were in charge of Dr. Eric Oldberg and Dr. Francis J. Gerty of the University of Illinois College of Medicine. Funds for nursing service were appropriated to and paid out by the State Department of Public Welfare. Funds for the physical plant, operation of maintenance, and for food service were appropriated to the department, but the services were provided by the university. Under the new plan, all activities and operations of the institute are vested in the University of Illinois. This change was recommended by the Shaefer Commission on State Reorganization and was concurred in by both the university and the State Department of Public Welfare. Dr. Oldberg will serve as director of the division of neurology and neurological surgery and Dr. Gerty as director of the division of psychiatry. The plan will simplify business procedures in the university, the department, and state fiscal offices. The purpose of the institute, as stated by the University of Illinois Board of Trustees, is to act as a liaison between the Illinois State Mental Health Service and the University of Illinois in a cooperative effort to improve the quality of the medical personnel dealing with the patients committed to their care, and to seek new and improved methods of treatment. The operation of the State Psychopathic Institute and the Pathological Laboratory, which are housed in the Illinois Neuropsychiatric Institute, will remain under the State Department of Public Welfare. Dr. Percival Bailey, professor of neurology and neurological surgery at the university, who previously served as executive officer of the institute for the State Department of Public Welfare, will be in charge of these activities for the department. The university, however, will assume the physical operation of the space used by these activities.

Chicago

Dr. Mote Moves to Philadelphia.—Dr. John R. Mote, research scientist responsible for much of the clinical development of corticotrophin (ACTH), has joined the Research and Development Laboratories of Smith, Kline and French Laboratories in Philadelphia as medical director. Formerly medical director and assistant general manager of Armour Laboratories, Dr. Mote will assume his new post with the Philadelphia pharmaceutical research and manufacturing firm in September.

Televised Postgraduate Conference.—The Third Annual Televised Postgraduate Conference in Obstetrics and Gynecology will be given Sept. 24-28 at Lewis Memorial Maternity Hospital. It will be presented by the department of obstetrics and gynecology of the Stritch School of Medicine of Loyola University under the chairmanship of Dr. Herbert E. Schmitz. The conference will include demonstrations of obstetrical procedures and gynecological surgery. The Radio Corporation of America will install television screens throughout the hospital to assure clear viewing and listening. There is no registration fee.

Appoint Dean of Indian Medical College.—Dr. Carroll L. Birch, professor of medicine at the University of Illinois, has been appointed dean of the Lady Hardinge Medical College for Women at New Delhi, India, for one year. The 100-year-old college, endowed by a British noblewoman, is the only Indian medical institution for the training of women. Dr. Birch's specific assignment will be to train a replacement to take over the duties of dean of the college. She also will prepare recommendations for the reorganization of teaching methods and curricula to raise school standards to meet those of modern medical educational institutions.

Research Grants.—Grants for research totalling more than $18,000 have recently been received by the Chicago Medical School. The Eli Lilly Research Laboratories gave $6,000 for work in experimental diabetes under the direction of Dr. Piero P. Foa, associate professor of physiology and pharmacology. The Chicago Heart Association, for the year July 1 to June 30, 1952, made grants of $2,000 and $6,800 for studies to be conducted under the direction of Dr. Aldo A. Luisada, program director of cardiology and assistant professor of medicine, and $4,000 for research directed by Dr. George J. Scheff, associate professor of microbiology.

Physicians are invited to send to this department items of news of general interest, for example, those relating to society activities, new hospitals, education and public health. Programs should be received at least three weeks before the date of meeting.

INDIANA

Ray Smith Dies.—Ray E. Smith, executive secretary of the Indiana State Medical Association since 1946, died Aug. 4 at his home in Indianapolis, aged 54, of a heart attack. A former newspaper man, and executive secretary to Gov. Henry F. Schricker in 1941, Mr. Smith had served as secretary of the Indianapolis Medical Society and served on a part-time basis for the state medical society at the same time. He was on the State Board of Tax Commission and was recently appointed to the Indiana Council of Mental Health. Last spring he was appointed to the lay public relations advisory committee of the American Medical Association. Mr. Smith was a past president of the Indiana Editorial Association and was a member of Sigma Delta Chi and the Indianapolis Press Club.

KENTUCKY

Historic Medical Gavel.—The Kentucky Surgical Society has received from its first president, Dr. Charles A. Vance, Lexington, a gavel made from wood having historical significance. The wood was collected by the late Dr. Joseph A. Stucky, Lexington, from the first building of the Transylvania Medical College in Lexington, from the home of Dr. Ephraim McDowell, Danville, and from a tree planted by Dr. McDowell in his backyard. Three gavels were made from these sources, one of which Dr. Vance has presented to the Southern Surgical Association and the other to the Southern Medical Association.

Hospital Honors Staff Veteran.—Dr. Michael J. Henry, Louisville, was recently honored by St. Joseph Infirmary at a dinner at the hospital in observance of his 50th anniversary of services. Dr. Henry has been closely connected with St. Joseph infirmary during most of the 50 years since his first job there in 1901 when at the age of 11 he was hired to do odd jobs about the hospital. He has been chairman of the City-County Health Board and president of the Jefferson County Medical Society. Last year he was named to the board of governors of the American College of Surgeons.

LOUISIANA

Center for the Hard of Hearing.—An anonymous gift of $5,000 will help establish a center for the hard of hearing at the Tulane University of Louisiana School of Medicine, New Orleans. Plans call for immediate construction of the center on the sixth floor of Hutchinson Memorial Medical Building. Establishment of a research program in rehabilitation of the deaf, and staffing of the center with research fellows in connection with this program, are planned for the future. Operating as a diagnostic clinic, the center will provide specific services not available in the area which Tulane serves.

MASSACHUSETTS

Personals.—Dr. Thomas S. Sexton has been appointed medical director of the Massachusetts Mutual Life Insurance Company of Springfield.——Dr. James L. Whittenberger, associate professor and head of the physiology department for four years, has been promoted to professor of physiology at the Harvard School of Public Health, Boston.

Appointments at Harvard.—Dr. Otto Krayer has been promoted to professor of pharmacology in the Harvard Medical School, Boston. As head of the department, he also will continue to instruct students and to do research. Dr. Krayer, who has been associate professor of comparative pharmacology since 1940, taught pharmacology at the American University of Beirut, Lebanon, before joining the staff at Harvard in 1937. He is a member of the Pharmacological Study Section of the United States Public Health Service and is on the editorial staff of "Pharmacological Reviews." He served as a member of the Unitarian Service Committee's medical mission to Czechoslovakia in 1946 and as chairman of the Unitarian mission to Germany in 1948. Other appointments include Dr. Jordi Folch-Pi, who becomes associate professor of biological chemistry at McLean Hospital in Waverley, continuing as chief of the biochemical laboratory there, and Dr. Nathan Bill Talbot, who becomes associate professor of pediatrics at Massachusetts General Hospital. Both doctors will carry on their research and teaching activities at the hospitals. Dr. Raymond Delacy Adams has been promoted from assistant professor to associate clinical professor of neurology and chief of the neurological service at Massachusetts General Hospital. He succeeds Dr. Charles S. Kubik in both positions. Dr. Kubik, who has now become lecturer on neuropathology, will continue his neurological research and practice at the hospital.

MICHIGAN

Continuation Courses.—Wayne University College of Medicine, Detroit, is giving postgraduate continuation courses Sept. 10 to Dec. 1, which are open to all qualifying persons. The subjects include microbiology, physiological chemistry, physiology and pharmacology, pathology, dermatology, internal medicine, surgery, and obstetrics. A postgraduate course in the early detection of cancer is to be given throughout the year. It will utilize the facilities of Yates Memorial Clinic, Detroit Receiving Hospital and the Veterans Administration Hospital in Dearborn. Registration should be made with the office of Postgraduate Medical Education at the College of Medicine, 1512 St. Antoine, before Sept. 8. Tuition for the last course is $50 per quarter, and the class is limited to 15.

MISSOURI

General Practitioners Study Club.—The following programs for 1951 featuring local physicians have been arranged by the General Practitioners Study Club of Greater St. Louis:

Sept. 19, Roy V. Boedeker, Practical Procedures in Gynecology for Treatment in the General Practitioners Office.
Oct. 17, James F. Dowd, Treatment of Common Injuries Resulting from Automobile Accidents.
Nov. 21, Adolph C. Lange, Ophthalmic Procedures for the General Practitioner.
Dec. 19, Leo J. LeBlanc, Office Management of Some of the Common Anorectal Diseases.

MONTANA

General Practitioners Meeting.—The Montana chapter of the American Academy of General Practice will hold its annual meeting at the Rainbow Hotel, Great Falls, Sept. 13. Following a dinner meeting at 7 p. m. and a business meeting, Dr. Mark H. Mothersill of the research staff of Eli Lilly and Company, Indianapolis, will speak on "General Practitioners and Allergic Reactions." All physicians are invited to attend. Dr. James A. Mueller, Lewistown, asks that all who plan to attend make their reservation with him not later than Sept. 1.

NEW YORK

Infectious Hepatitis.—Dr. Milton Tully, New York State Department of Health, has reported an outbreak of infectious hepatitis in rural Chemung County. Twenty cases occurred from Jan. 11 to July 8, half of them in June. Cases were found principally in school children and were linked by personal contact.

Bulbar Poliomyelitis Center.—A new center for the clinical study of bulbar and respiratory poliomyelitis will be established at the University of Buffalo's Chronic Disease Research Institute under a grant from the National Foundation for Infantile Paralysis. The respirator center, one of four of its kind to be set up in the United States with March of Dimes funds, will have a capacity of at least ten respirators and will engage in research and clinical investigations on the recovery and rehabilitation of serious poliomyelitis cases. In a ceremony at the institute July 25 the foundation presented to the university its first check of $41,048 for the project. The medical school will immediately begin to prepare, equip, and staff the unit. Dr. Walter T. Zimdahl, medical director of the institute, will co-ordinate the activities of the center with those of the institute. The National Foundation said it selected Buffalo as the location of the new center on the recommendation of the New York State Coordinating Council of Poliomyelitis. Other respirator units sponsored by the foundation are now operating at the Children's Medical Center of Boston, Wellesley Hills, Mass.; at Jefferson Davis Hospital, Houston, Texas, and at University Hospital, Ann Arbor, Mich. In the joint announcement issued by the National Foundation and the University of Buffalo, the purpose of the center was described as three-fold:

1. To determine through special patient evaluation methods and by newer clinical investigative techniques and equipment how best to wean patients from iron lungs and enable them to be cared for at home, with the provision of physical therapists, occupational therapists, and psychological and medical social services to assist in the maximal rehabilitation of patients.

2. To provide opportunity for special study for doctors, nurses, and physical therapists, and more intensive training for resident physicians and medical students in the field of respiratory involvement in poliomyelitis, and to provide consultation and assistive services to hospitals in upstate New York.

3. To evaluate equipment used in diagnosis and treatment, and to cooperate in efforts to develop improvements.

New York City

Hillman Health Center to Expand.—This health center of the Amalgamated Clothing Workers, C. I. O., which opened four months ago (THE JOURNAL, May 26, p. 384), must be redesigned to permit the handling of a larger number of patients. The six-story building will be enlarged to include installation of advanced x-ray equipment and a research laboratory for studies in arteriosclerosis. Each week about 1,800 clothing workers go to the center for examination and treatment.

State University Appointments.—James O. Pinkston, Ph.D., has been appointed lecturer in the department of physiology at the State University of New York College of Medicine at New York City. From 1943 to 1950 Dr. Pinkston was dean of the medical division, American University of Beirut, Lebanon, where he had previously served as associate professor and chairman of the department of pharmacology and later as professor and chairman of the department of physiology. Recently he was on the staff of Cornell University Medical College.

OREGON

Rural Health Council.—Twenty-six state-wide organizations concerned with the health of the rural population of the state have organized the Oregon Health Council. Objectives of the council are to bring together representatives of state organizations and agencies which are now conducting programs affecting rural health for the purpose of discussing and exchanging views, and developing recommendations for improving rural health, and to encourage and stimulate the establishment of county or community health councils or committees. Chairman of the council is Dr. Howard A. Wells, Redmond, chairman of the Oregon State Medical Society committee on rural medical service; vice-chairman, Mrs. Amy S. Larkin, Portland, of the farm home administration of the U. S. Department of Agriculture, and secretary, Mrs. Mable C. Mack, Corvallis, Oregon State College Extension Service.

PENNSYLVANIA

Psychiatric Speaker's Bureau.—The Pennsylvania Psychiatric Society has established a speaker's bureau which will furnish, on request, psychiatrists to speak before lay and professional groups. Requests should be addressed to Dr. Stanley W. Conrad, 1822 Spruce St., Philadelphia.

Heart Association Fellowship.—The First Fellowship of the Heart Association of Southeastern Pennsylvania, amounting to an annual grant of $3,000 a year, has been awarded to Dr. Arthur Goldman, Philadelphia, who will work under the jurisdiction of Drs. Thomas M. McMillan and Samuel Bellet, Philadelphia General Hospital.

The Philadelphia Heart Association has changed its name to the Heart Association of Southeastern Pennsylvania because it serves Bucks, Chester, Delaware, and Montgomery counties as well as Philadelphia County. The officers are Dr. Lauritz S. Ylvisaker, president; Dr. Warner F. Haldeman, vice president; Mr. William T. Carter, treasurer, and Dr. Paul H. Parker, secretary.

Philadelphia

New Type Contagious Institution.—In July Philadelphia Hospital for Contagious Diseases opened facilities that will enable the simultaneous hospitalization of various types of contagious diseases. Patients will be quartered in units of two and five beds, each having its own bathing and lavatory facilities, and each with a combined heating and ventilating system capable of changing the air at the rate of 750 cubic feet per minute. A total of 54 patients suffering from various diseases will be cared for. At any time the ward door is opened the lower air pressure inside pulls the air into the ward, thus preventing any contaminated air from finding its way into the corridor. Dedication of this West Wing Observation Building, as it is to be known, was held July 10 with Dr. Rufus S. Reeves, director of public health, presiding.

WASHINGTON

Report of Plague Specimens.—Dr. Vernon B. Link, Western Communicable Disease Center Laboratory, has reported the following specimens obtained in Lincoln County were proved positive for plague: (1) Specimen No. 51-WB-26, consisting of 39 fleas (Megabothris clantoni clantoni) from 43 sagebrush voles (Lagurus curtatus) which were trapped 12 miles southeast of Wilbur, (9.6 miles south on State Highway 4B and then 5 miles east on county road) on July 5; and (2) Specimen No. 51-WB-27, consisting of 137 fleas (61 Thrassis gladiolis johnsoni and 76 Megabothris clantoni clantoni) from 38 Lagurus curtatus, trapped 17 miles north of Odessa, on State Highway No. 4B, on July 7.

GENERAL

Society Election.—At its annual meeting the American Medical Women's Association, Inc., elected Dr. Amey Chappell, Atlanta, president; Dr. Evangeline E. Stenhouse, Chicago, president-elect; Dr. Adelaide Romaine, New York, treasurer; Dr. Esther C. Marting, Cincinnati, recording secretary, and Dr. Irma C. Henderson-Smathers, Asheville, N. C., corresponding secretary.

Dr. Faulkner Heads Specialty Board.—Dr. Robert L. Faulkner, Cleveland, has been elected secretary-treasurer of the American Board of Obstetrics and Gynecology, to succeed the late Dr. Paul Titus. Dr. Lawrence M. Randall, Rochester, Minn., has been elected assistant secretary, and Dr. Herbert E. Schmitz, Chicago, a director of the board. Effective Aug. 10, the office of the board will be located at 2105 Adelbert Road, Cleveland 6. All correspondence should be directed to Dr. Faulkner at this address.

Dr. Hussey Named to Editorial Staff.—Dr. Hugh H. Hussey, associate professor of medicine at Georgetown University, Washington, D. C., has been appointed associate editor of *GP*, published by the American Academy of General Practice. Dr. Hussey has for years been associate editor of the *Medical Annals of the District of Columbia*. He is author of the section on "Supportive and Symptomatic Therapy" in the textbook "Fundamentals of Internal Medicine" now in its third edition. He carries on a consultation practice in Washington and is a member of the staff of Gallinger Municipal Hospital and the Georgetown University Hospital and is senior consultant on vascular diseases at the Mount Alto Veterans Hospital.

Tuberculosis Service Workers' Course.—The National Tuberculosis Association will conduct a general training course for new professional workers at Roycroft Inn, East Aurora, New York, Sept. 4-29. The course is open to staff members of state and local tuberculosis associations who have had at least one month of experience and not more than 10 months, and to college graduates seeking careers in the tuberculosis field. The course is designed to acquaint new workers with basic concepts and techniques in voluntary tuberculosis association work. Field trips will supplement the formal curriculum, which includes lectures on all phases of social service work for the tuberculous.

Gold Heart Awards.—These awards for outstanding contributions to cardiovascular medicine or the American Heart Association were presented at the association's annual dinner in Atlantic City in June to Drs. James B. Herrick, Frank N. Wilson, and Harold M. Marvin. Dr. Herrick, emeritus professor of medicine at the University of Chicago and the University of Illinois, was honored for his work on coronary thrombosis. Dr. Wilson, professor of medicine at the University of Michigan,

received the award for his investigations in the field of electrocardiography. Dr. Marvin, New Haven, Conn., received the Gold Heart for his many years of service to the association, including the presidency in 1949-1950.

Prevalence of Poliomyelitis.—According to the National Office of Vital Statistics, the following number of reported cases of poliomyelitis occurred in the United States in the weeks ended as indicated:

Area	Aug. 4, 1951	Aug. 5, 1950
New England States		
Maine	1	1
New Hampshire	9	..
Vermont	2	..
Massachusetts	19	9
Rhode Island	..	2
Connecticut	12	7
Middle Atlantic States		
New York	65	95
New Jersey	17	24
Pennsylvania	35	49
East North Central States		
Ohio	37	44
Indiana	10	14
Illinois	81	77
Michigan	73	41
Wisconsin	34	23
West North Central States		
Minnesota	28	13
Iowa	14	46
Missouri	12	15
North Dakota	5	1
South Dakota	5	2
Nebraska	23	19
Kansas	36	29
South Atlantic States		
Delaware	1	1
Maryland	1	11
District of Columbia	4	15
Virginia	11	88
West Virginia	9	19
North Carolina	21	41
South Carolina	3	26
Georgia	49	30
Florida	16	12
East South Central States		
Kentucky	15	42
Tennessee	30	45
Alabama	43	12
Mississippi	38	18
West South Central States		
Arkansas	23	11
Louisiana	34	29
Oklahoma	52	35
Texas	92	122
Mountain States		
Montana	2	3
Idaho	..	2
Wyoming	9	3
Colorado	71	7
New Mexico	2	6
Arizona	3	2
Utah	23	2
Nevada	1	..
Pacific States		
Washington	8	9
Oregon	14	7
California	110	76
Territories and Possessions		
Alaska	..	..
Hawaii	1	2
United States	1,294	1,187

Mississippi Valley Society Meetings.—The Mississippi Valley Medical Society will hold its annual meeting at the Hotel Pere Marquette, Peoria, Ill., Sept. 19-21, under the presidency of Dr. Ralph McReynolds, Quincy, Ill. Thirty-two scientific papers will be presented. The visiting speaker will be Dr. Oscar C. Hansen-Pruss, professor of medicine at The Duke University, Durham, N. C. There will be round table luncheons Thursday and Friday. Speakers at the evening fellowship hour, banquet, and entertainment on Thursday will be Drs. C. Paul White, Kewanee, Ill.; C. Edgar Virden, Kansas City, Mo.; Donald C. Conzett, Dubuque, Iowa, and Alexander Baird, Ph.D., Peoria, Ill. There is no registration fee.

The American Medical Writers' Association will meet at the hotel on Sept. 19 under the presidency of Dr. Julius Jensen, St. Louis. The session, beginning in the afternoon, will be addressed by Dr. Richard M. Hewitt, head of the section of publications, Mayo Clinic, Rochester, Minn. Other speakers will be Miss Marguerite Stadelhofer, managing editor of C. V. Mosby Company, medical publishers, who will speak on "The Making of Medical Books." At the evening session Dr. Ralph H. Major, professor of medicine and of the history of medicine at the University of Kansas City, Kansas City, Mo., will speak on "Some of the Great Books of Medicine."

American Cancer Society Fellowship Awards.—Three Chicago scientists have been awarded fellowships for cancer research totaling $13,000 for studies of growth processes in plants and animals. Dr. John M. Marshall Jr. will study for one year at the Carlsberg Laboratory in Copenhagen, Denmark, where he will investigate the method by which plant constituents are isolated and purified. Robert C. Barnett, Ph.D., and Richard M. Klein, Ph.D., both of the University of Chicago, will conduct their research there. These scientists were selected from applicants in all parts of the country and were recommended for American Cancer Society fellowships by the Committee on Growth of the National Research Council.

FOREIGN

Communicable Diseases.—According to reports received by the regional office of the World Health Organization, an outbreak of poliomyelitis has been in progress in Mexico City since the first part of May. Since then over 400 cases have been reported. The peak of the outbreak apparently was reached in the week ending June 23 when there were 100 new cases. For the weeks ending June 30 and July 7, there were 54 and 40 cases, respectively, reported. For the week ending July 14, there were 12 new cases in the city of Birmingham, England.

A high prevalence of diphtheria continues in Rio de Janeiro, Brazil, 86 cases with nine deaths being reported for the three-week period ending on July 7. In Santiago, Chile, 45 cases and one death occurred in the week ending July 7; and in San Jose, Costa Rica, there were 10 cases in the second week of July. In Bangkok, Thailand, and in Singapore, 20 and eight cases, respectively, were reported for one week early in July. According to a World Health Organization report, there were 1,419 cases of brucellosis in Italy in May as compared with 1,172 in April. During the first two weeks of June 37 cases were reported on the Island of Malta and Gozo. In the cities of Spain with less than 20,000 population, there was a total of 618 cases with five deaths reported in a seven-week period ending April 14. In cities with more than 20,000 population only 17 cases were reported in the same period.

MEETINGS

AMBULATORY FRACTURE ASSOCIATION, Arrowhead Springs Hotel, San Bernardino, Calif., Oct. 8-11. Dr. H. W. Wellmerling, 626 Griesheim Bldg., Bloomington, Ill., Secretary.

AMERICAN ASSOCIATION OF OBSTETRICIANS, GYNECOLOGISTS AND ABDOMINAL SURGEONS, The Homestead, Hot Springs, Va., Sept. 6-8. Dr. William F. Mengert, 2211 Oak Lawn Ave., Dallas, Texas, Secretary.

AMERICAN CONGRESS OF PHYSICAL MEDICINE, Shirley-Savoy Hotel, Denver, Sept. 4-8. Dr. Walter J. Zeiter, 2020 E. 93d St., Cleveland 6, Executive Director.

AMERICAN HOSPITAL ASSOCIATION, St. Louis, Sept. 17-20. Mr. George Bugbee, 18 E. Division St., Chicago 10, Executive Director.

AMERICAN ROENTGEN RAY SOCIETY, Washington, D. C., Sept. 25-28. Dr. Barton R. Young, Germantown Hospital, Philadelphia 44, Secretary.

AMERICAN SOCIETY OF MAXILLOFACIAL SURGEONS, Drake Hotel, Chicago, Sept. 23-26. Dr. Casper M. Epsteen, 25 E. Washington St., Chicago 2, Secretary.

ASSOCIATION OF MILITARY SURGEONS OF THE UNITED STATES, Palmer House, Chicago, Oct. 8-10. Colonel James M. Phalen, Armed Forces Institute of Pathology, Washington 25, D. C., Secretary.

COLORADO STATE MEDICAL SOCIETY, Denver, Sept. 18-21. Mr. Harvey T. Sethman, 1612 Tremont Place, Denver 2, Executive Secretary.

DELAWARE, MEDICAL SOCIETY OF, Wilmington, Oct. 8-10. Dr. Andrew H. Gehret, 1007 Park Place, Wilmington, Secretary.

DISTRICT OF COLUMBIA, MEDICAL SOCIETY OF, Hotel Statler, Washington, Oct. 1-3. Mr. Theodore Wiprud, 1718 M St. N.W., Washington, Secretary.

KANSAS CITY SOUTHWEST CLINICAL SOCIETY, Municipal Auditorium, Kansas City, Mo., Oct. 1-4. Dr. Maxwell G. Berry, 630 Shukert Bldg., Kansas City, Mo., Director of Clinics.

KENTUCKY STATE MEDICAL ASSOCIATION, Columbian Auditorium, Louisville, Oct. 2-5. Dr. Bruce Underwood, 620 S. Third St., Louisville 2, Secretary.

MICHIGAN STATE MEDICAL SOCIETY, Grand Rapids, Sept. 26-28. Dr. L. Fernald Foster, 606 Townsend St., Lansing 15, Secretary.

Mississippi Valley Medical Society, Peoria, Ill., Sept. 19-21. Dr. Harold Swanberg, 510 Maine St., Quincy, Ill., Secretary.
Montana Medical Association, Great Falls, Sept. 13-16. Mr. L. R. Hegland, 104 N. Broadway, Billings, Executive Secretary.
Nashville Postgraduate Medical Assembly, Maxwell House, Nashville, Tenn., Oct. 3-5. Dr. W. G. Kennon Jr., 647 Doctors Bldg., Nashville 3, Secretary.
National Medical Association, Philadelphia, Aug. 20-24. Dr. John T. Givens, 1108 Church St., Norfolk 10, Va., General Secretary.
Neurosurgical Society of America, Sun Valley, Idaho, Sept. 12-14. Dr. C. D. Hawkes, 22 N. Manassas St., Memphis 5, Tenn., Secretary.
Nevada State Medical Association, Riverside Hotel, Reno, Sept. 13-15. Dr. Wesley W. Hall, 307 W. Sixth St., Reno, Secretary.
New Hampshire Medical Society, Equinox House, Manchester, Vermont, Sept. 30-Oct. 2. Dr. Deering G. Smith, 44 Chester St., Nashua, Secretary.
Northeastern Section, Southeastern Surgical Congress, Lord Baltimore Hotel, Baltimore, Sept. 13-15. Dr. W. Raymond McKenzie, Medical Arts Bldg., Baltimore 1, Secretary.
North Texas-Southern Oklahoma Fall Clinical Conference, Wichita Falls, Texas, Sept. 19. Dr. W. L. Powers, 517 Hamilton Bldg., Wichita Falls, Chairman.
Oregon State Medical Society, Masonic Temple, Portland, Oct. 10-13. Dr. Robert F. Miller, 833 S.W. Eleventh Ave., Portland 5, Secretary.
Pennsylvania, Medical Society of the State of, Pittsburgh, Sept. 16-20. Dr. Walter F. Donaldson, 500 Penn Ave., Pittsburgh 22, Secretary.
Reno Surgical Society Conference, Riverside Hotel, Reno, Nev., Aug. 23-25. Dr. Olin C. Moulton, 130 N. Virginia St., Reno, Nev., Chairman.
Southwestern Surgical Congress, Hotel Jefferson, St. Louis, Sept. 24-26. Dr. C. R. Rountree, 1227 Classen St., Oklahoma City 3, Secretary.
Texas Academy of General Practice, Shamrock Hotel, Houston, Texas, Sept. 10-11. Dr. B. H. Bayer, 104 E. 20th St., Houston 8, Secretary.
U. S. Chapter, International College of Surgeons, Palmer House, Chicago, Sept. 10-14. Dr. Arnold S. Jackson, 1516 Lake Shore Drive, Chicago, Executive Secretary.
Utah State Medical Association, Salt Lake City, Sept. 13-15. Dr. T. C. Weggeland, 42 S. Fifth East St., Salt Lake City, Secretary.
Vermont State Medical Society, Equinox House, Manchester, Sept. 30-Oct. 2. Dr. James P. Hammond, 542 Main St., Bennington, Secretary.
Virginia, Medical Society of, Virginia Beach, Oct. 7-10. Mr. Richard H. Nash, 1105 W. Franklin St., Richmond, Executive Secretary.
Washington State Medical Association, Olympic Hotel, Seattle, Sept. 9-12. Dr. James W. Haviland, 338 White-Henry-Stuart Bldg., Seattle, Secretary.
Western Society of Electro-Encephalography, Olympic Hotel, Seattle, Sept. 7-8. Dr. Nicholas A. Bercel, 450 N. Bedford Drive, Beverly Hills, Calif., Secretary.
Wisconsin, State Medical Society of, Hotel Schroeder and Milwaukee Auditorium, Milwaukee, Oct. 1-3. Mr. Charles H. Crownhart, 704 E. Gorham St., Madison 3, Secretary.
Wyoming State Medical Society, Rock Springs, Sept. 27-29. Dr. Glenn W. Koford, 2020 Carey Ave., Cheyenne, Secretary.

INTERNATIONAL

European Congress on Rheumatism, Barcelona, Spain, Sept. 24-27. Dr. Gunnar Edström, Lund, Sweden, Secretary.
General Practitioners Study Club International, Rome, Italy, Sept. 12-15. Dr. John O'Connell, 10300 Lackland Road, St. Louis 14, Mo., U. S. A., President.
International Anesthesia Research Society, London, England, Sept. 3-7. Mr. R. W. Cope, University College Hospital, London W.C.1, England.
International Association of Allergists, Zurich, Switzerland, Sept. 23-29. Prof. A. S. Grumbach, Hygiene Institut der Universitaet Zurich, Gloriastr. 32, Zurich, Switzerland.
International Congress of Anesthesiology, Nursing School of the Salpiettre, 47 Boul de l'Hospital, Paris, France, Sept. 20-22. 12 rue de Seine, Paris 6e, France, Secretariat.
International Congress on Entomology, Amsterdam, Holland, Aug. 17-24. Dr. J. de Wilde, 136 Rapenburgerstraat, Amsterdam, Secretary.
International Congress on Industrial Medicine, Instituto Superior Técnico, Lisbon, Portugal, Sept. 9-15. Dr. Luis Guerreiro, Instituto Nacional do Trabalho e Previdência, Praça do Comercio, Lisbon, Secretary General.
International Congress on Mental Health, Mexico City, Mexico, Dec. 11-19. Mrs. Grace E. O'Neill, Division of World Affairs, National Association on Mental Health, 1790 Broadway, New York 19, N. Y.
International Congress of Physical Medicine, London, England, July 14-19. Dr. A. C. Boyle, 45, Lincoln's Inn Fields, London, W.C.2, England, Honorary Secretary.
International Gerontological Congress, Hotel Jefferson, St. Louis, Mo., U. S. A., Sept. 9-14. Dr. John E. Kirk, 5600 Arsenal Street, St. Louis 9, Mo., Chairman, Program Committee.
International Poliomyelitis Congress, Copenhagen, Denmark, Sept. 3-7. Prof. Dr. Niels Bohr, Statens Seruminstitut, 80 Amager Blvd., Copenhagen S., Denmark, President.
International Society of Surgery, Paris, France, Sept. 24-29. Dr. L. Dejardin, 141, rue Belliard, Brussels, Belgium, Secretary General.
International Society for the Welfare of Cripples, Fifth World Congress, Stockholm, Sweden, Sept. 9-14. Mr. Donald V. Wilson, 54 E. 64th St., New York 21, N. Y., U. S. A., Executive Director.
Pan Pacific Surgical Association Congress, Honolulu, Hawaii, Nov. 7-19. Dr. Forrest J. Pinkerton, Suite 7, Young Bldg., Honolulu, Hawaii, President.
World Confederation for Physicial Therapy, Copenhagen, Denmark, Sept. 7-8.
World Medical Association, Stockholm, Sweden, Sept. 15-21. Dr. Louis H. Bauer, 2 E. 103d St., New York 29, N. Y., U. S. A., Secretary General.

EXAMINATIONS AND LICENSURE

BOARDS OF MEDICAL EXAMINERS

Alabama: *Examination.* Montgomery, June 24-26, 1952. Sec., Dr. D. G. Gill, 537 Dexter Ave., Montgomery 4.
Arkansas:* *Regular.* Little Rock, Nov. 8-9. Sec., Dr. Joe Verser, Harrisburg.
California: *Written.* San Francisco, Aug. 20-23. *Oral.* Los Angeles, August 18. *Oral and Clinical Examination for Foreign Medical School Graduates.* Los Angeles, August 19. Sec., Dr. Frederick N. Scatena, 1020 N St., Sacramento 14.
Colorado:* *Reciprocity.* Denver, Oct. 9. Final date for filing application is Sept. 24. Ex. Sec., Mrs. Beulah H. Hudgens, 831 Republic Bldg., Denver 2.
Georgia: Atlanta, June. August, Oct. 9-11. Sec., Mr. R. C. Coleman, 111 State Capitol, Atlanta.
Guam: The Commission on Licensure will meet whenever a candidate appears or submits his credentials. Ex. Sec., Dr. John Y. Battenfield, Agana.
Illinois: Chicago, Oct. 9-11. Superintendent of Registration, Mr. Charles F. Kervin, Capitol Building, Springfield.
Indiana: Indianapolis, June 1952. Sec., Dr. Paul R. Tindall, 1138 K of P Bldg., Indianapolis 4.
Kansas: Topeka, Dec. 12-13. Sec., Dr. O. W. Davidson, 772 New Brotherhood Bldg., Kansas City.
Kentucky: *Examination.* Louisville, Dec. 11-13. Sec., Dr. Bruce Underwood, 620 S. 3rd St., Louisville.
Maryland: *Examination.* Baltimore, Dec. 11-14. Sec., Dr. Lewis P. Gundry, 1215 Cathedral St., Baltimore.
Michigan:* *Examination.* Lansing, Oct. 10-12. Sec., Dr. J. Earl McIntyre, 202-4 Hollister Bldg., Lansing.,
Minnesota: * Minneapolis, Oct. 16-18. Sec., Dr. Julian F. DuBois, 230 Lowry Medical Arts Bldg., St. Paul 2.
Mississippi: *Reciprocity.* Jackson, December. Asst. Sec., Dr. R. N. Whitfield, Old Capitol Bldg., Jackson.
New Hampshire: Concord, Sept. 12-13. Sec., Dr. John Samuel Wheeler, Room 107 State House, Concord.
New Jersey: *Examination.* Trenton, Oct. 16-19. Sec., Dr. E. S. Hallinger, 28 W. State St., Trenton.
New Mexico:* Santa Fe, Oct. 8-9. Sec., Dr. Charles J. McGoey, Coronado Bldg., Santa Fe.
New York: *Examination.* Albany, Buffalo, New York and Syracuse, Oct. 31-Nov. 3. Sec., Dr. Jacob L. Lochner, Jr., 23 S. Pearl St., Albany.
North Carolina: Asheville, Oct. 7-8. Sec., Dr. Joseph J. Combs, Professional Bldg., Raleigh.
Ohio: *Examination.* Columbus, December. *Reciprocity.* Columbus, Oct. 2. Sec., Dr. H. M. Platter, 21 W. Broad St., Columbus 15.
Oregon:* *Reciprocity.* Portland, Oct. 12-13. Ex. Sec., Mr. Howard I. Bobbitt, 609 Failing Bldg., Portland.
Puerto Rico: *Examination.* Santurce, Sept. 4-7. Sec., Mr. Luis Cueto Coll, Box 3717, Santurce.
South Carolina: *Reciprocity.* Columbia, Sept. 10 and Oct. 1. Sec., Dr. N. B. Heyward, 1329 Blanding St., Columbia.
South Dakota:* *Examination.* Sioux Falls, Jan. 15-16. Ex. Sec., Mr. John C. Foster, 300 First National Bank Bldg., Sioux Falls.
Virgin Islands: Charlotte Amalie, Nov. 7-8. Sec., Dr. Earle M. Rice, St. Thomas.
Wyoming: *Examination.* Cheyenne, Oct. 1. Sec., Dr. Franklin D. Yoder, State Capitol, Cheyenne.

BOARDS OF EXAMINERS IN THE BASIC SCIENCES

Arizona: *Examination.* Tucson, Sept. 18. Sec., Mr. Francis A. Roy, 423 Liberal Arts Bldg., University of Arizona, Tucson.
Connecticut: *Examination.* New Haven, Oct. 13. Exec. Asst., Miss Mary G. Reynolds, State Board of Healing Arts, 110 Whitney Avenue, New Haven 10.
District of Columbia: *Examination.* Washington, Oct. 22-23. Sec., Dr. Daniel L. Seckinger, 4130 E. Municipal Bldg., Washington.
Michigan: Detroit and Ann Arbor, Oct. 12-13. *Examination.* Sec., Miss Eloise LeBeau, 501 W. Michigan Ave., Lansing 15.
Minnesota: *Examination.* Minneapolis, Oct. 2-3. Sec., Dr. Raymond N. Bieter, 105 Millard Hall, University of Minnesota, Minneapolis.
Nebraska: *Examination.* Omaha, Oct. 2-3. Director, Bureau of Examining Boards, Mr. Oscar F. Humble, Room 1009, State Capitol Bldg., Lincoln 9.
Oklahoma: Oklahoma City, Sept. 14. Sec., Dr. Frank C. Gallaher, 813 Braniff Bldg., Oklahoma City 4.
Oregon: *Examination.* Portland, Sept. 8 and Dec. 1. Sec., Dr. Charles D. Byrne, State Board of Higher Education, University of Oregon, Eugene.
South Dakota: *Examination.* Vermillion, Dec. 7-8. Sec., Dr. Gregg M. Evans, 310 E. 15th St., Yankton.
Texas: *Examination.* Austin, October. Chief Clerk, Mr. B. H. Kirk, 306 Nalle Bldg., Austin.
Wisconsin: *Examination.* Madison, Sept. 22. Sec., Mr. W. H. Barber, Ripon College, Ripon.

* Basic Science Certificate required.

DEATHS

MacNider, William de Berniere ✦ Chapel Hill, N. C.; born in Chapel Hill June 25, 1881; University of North Carolina School of Medicine, Chapel Hill, 1903; Kenan research professor of pharmacology, emeritus, at his alma mater, which he had joined in 1905 as Kenan professor of pharmacology, becoming Kenan research professor of pharmacology in 1920, and serving as dean from 1937 to 1940; the William B. MacNider Award was established at the school in 1951 for public commendation of a sophomore medical student possessing the traits of character typified by Dr. MacNider during his 50 years as teacher and physician in the university; at various times special lecturer at Columbia University in New York, George Washington University School of Medicine, Washington, D. C., Medical College of Virginia, Richmond, Mayo Foundation, Rochester, Minn., and The Duke University School of Medicine, Durham, N. C.; formerly Harvey Society lecturer; consultant on gerontology, National Institutes of Health, Bethesda, Md.; chairman, Section on Pharmacology and Therapeutics of the American Medical Association, 1926-1927; member of the National Board of Medical Examiners and chairman of the examination committee, member of the National Research Council committee on nutritional aspects of ageing, the committee on cellular physiology, executive committee of the medical division; chairman of the American Division of the International Club for Research on Ageing; fellow of the American Association for the Advancement of Science, American Academy of Arts and Sciences, and the American College of Physicians; member of the American Society for Pharmacology and Experimental Therapeutics (president from 1932 to 1934, and member of the council), president of the Medical Society of the State of North Carolina (1925-1926), member of the American Physiological Society, American Association of Pathologists and Bacteriologists, American Association of Biological Chemists, Society of Experimental Biology and Medicine (president 1941-1942), American Association of University Professors, American Society for Experimental Pathology, Association of American Physicians, American Society of Anethetists, National Anaesthesia Research Society and member of the research committee, International Anaesthesia Research Society (president 1934-1935), American Association for the History of Medicine, North Carolina Academy of Science, National Academy of Sciences, New York Academy of Sciences, American Society of Naturalists, American Philosophical Society, and the Harvey Society; honorary member of the Pathological Society of Great Britain and Ireland; associate foreign member of the British Physiological Society; chairman of the council of the Gerontological Society; member of Sigma Xi, Sigmu Nu, Phi Beta Kappa, and Alpha Omega Alpha; was awarded the George M. Kober Medal in 1941 by the Association of American Physicians for research on injury to tissue cells, the New York Academy of Medicine's Edward M. Gibbs Memorial Prize in 1931; two years later was presented with the Southern Medical Association research medal; on the editorial board of the *Quarterly Journal of Studies on Alcohol;* contributor of articles to medical and biological journals; received the honorary degrees of doctor of science from the Medical College of Virginia in 1933 and doctor of laws from Davidson College in 1934; on the honorary staff of Watts Hospital in Durham, where he died May 31, aged 70, of congestive heart disease.

Morrison, Richard John, Santa Monica, Calif.; born in Glasgow, Scotland, June 22, 1880; Loyola University School of Medicine, Chicago, 1918; member of the American Medical Association; past president of Santa Monica Medical Society; president of the Marion Davies Child Guidance Clinic; chairman of the Santa Monica Welfare Clinic staff; served on the staffs of the Los Angeles County Hospital in Los Angeles and Santa Monica Hospital; past president of the Lions Club; served with the Salvation Army in Java two years during his early medical career; died May 28, aged 70, of coronary occlusion.

Maybaum, Jacob Louis ✦ New York; Cornell University Medical College, New York, 1905; specialist certified by the American Board of Otolaryngology; member of the American Academy of Ophthalmology and Otolaryngology, American Laryngological, Rhinological and Otological Society, and the American Otological Society; fellow of the American College of Surgeons; consulting otologist, Mount Sinai Hospital, where he died May 31, aged 67, of congestive heart failure.

Morrow, Roy Darlington ✦ Connersville, Ind.; Indiana Medical College, School of Medicine of Purdue University, Indianapolis, 1907; past president of the Fayette-Franklin Counties Medical Society; served during World War I; on the staff of Fayette Memorial Hospital; died May 30, aged 67, of coronary occlusion.

Potter, La Forest, Haverford, Pa.; Boston University School of Medicine, 1884; died in St. Agnes Hospital, Philadelphia, recently, aged 85, of carcinoma of the esophagus.

Richardson, Elbert Leech, Louisville, Miss.; University of Nashville (Tenn.) Medical Department, 1907; member of the American Medical Association; for many years county health officer; past president of the East Mississippi Medical Association; died May 26, aged 69.

Rosamond, John Stribling, West, Miss.; Memphis (Tenn.) Hospital Medical College, 1901; member of the American Medical Association; medical examiner for Selective Service during World War I; for many years president of the county board of education, county health officer, and local surgeon for the Illinois Central Railroad; affiliated with Holmes County Community Hospital in Lexington and the District Two Community Hospital in Durant; died May 29, aged 77, of injuries received in an automobile accident.

Smith, Charles Edgar, Beloit, Wis.; Northwestern University Medical School, Chicago, 1901; past president of the hospital staff, Beloit Hospital; died May 21, aged 78, of coronary thrombosis and arteriosclerotic heart disease.

Southworth, Hamilton Munn, Owensburg, Ind.; Albany (N. Y.) Medical College, 1905; served during World War I; died May 26, aged 70, of cerebral hemorrhage.

Stocks, John Carter, Bountiful, Utah; Northwestern University Medical School, Chicago, 1911; member of the American Medical Association; formerly mayor of Bountiful and city councilman; died in a hospital in Ogden May 28, aged 72, of coronary occlusion.

Strong, Sneed, Dallas, Texas; Missouri Medical College, St. Louis, 1891; served during World War I; formerly affiliated with the Veterans Administration; died in the Veterans Administration Hospital, Waco, May 12, aged 86, of arteriosclerosis.

Waldrep, Archie C., Red Bay, Ala.; University of Louisville (Ky.) Medical Department, 1893; member of the American Medical Association; died May 26, aged 82, of coronary thrombosis.

Walters, John K., Mooresburg, Tenn. (licensed in Tennessee in 1892); died in East Tennessee Baptist Hospital, Knoxville, May 25, aged 86, of coronary thrombosis.

Whitacre, John Clifford ✦ St. Paul; University of Minnesota College of Medicine and Surgery, Minneapolis, 1901; died May 28, aged 74, of coronary thrombosis.

Wight, William G. ✦ Yale, Mich.; Trinity Medical College, Toronto, Canada, 1890; served as mayor of Yale and as president of the Yale Bank; affiliated with Yale Hospital and the Port Huron (Mich.) Hospital; died in Women's Hospital, Detroit, May 28, aged 83, of myocardial infarction.

Wiley, Edwin Hardin ✦ Los Angeles; Northwestern University Medical School, Chicago, 1901; fellow of the American College of Surgeons; on the consulting staff of St. Joseph's Hospital in Burbank, Calif., and on the staff of California Hospital, where he died May 28, aged 73, of carcinoma of the pancreas with metastases.

✦ Indicates Fellow of the American Medical Associati( ).

GOVERNMENT SERVICES

AIR EVACUATION OF PATIENTS IN KOREA

Korea (Fig. 1).—This is the beginning of an Air Force medical air evacuation. These casualties on litters from Korea will be flown to Japan for screening. The patients are being carried aboard a Douglas C-54 "Skymaster" of the USAF Troop Carrier Command.

Tachikawa, Japan (Fig. 2).—Just in from an air evacuation flight from Korea, a flight nurse and a medical technician carry their medical gear from the C-54 Douglas "Skymaster" that acted as a flying hospital. Combat casualties, picked up at a Korean airstrip, have been already unloaded into ambulances and are on their way to service hospitals for treatment and screening.

Korea (Fig. 3).—This hospital on the edge of an operational airstrip is 30 minutes from the front lines. Medical technicians are readying combat casualties for medical air evacuation flights to Japan, the first leg of the air evacuation trans-Pacific crossing of the Pacific Airlift.

Tachikawa, Japan (Fig. 4).—Japanese civilian employees carry a Korean combat casualty from an Air Force C-54 Douglas "Skymaster" to a waiting ambulance for hospitalization. Air evacuation of wounded has been a major development of the Korean conflict.

Korea (Fig. 5).—The combat casualties start home by air from a dusty Korean airstrip. Medical technicians like these checking litter straps in this Air Force C-54 "Skymaster" transport, will tend the wounded through their flight to Japan and later, on the Pacific Airlift, to Continental United States.

ARMY

Malaria in Veterans Who Have Served in Korea.[1]—There are now returning from Korea many members of the Armed Forces who, while in combat, were exposed to the disease hazards of that country. The most common diseases encountered there have been bacillary dysentery, malaria, typhoid fever, typhus fever, infectious hepatitis, Japanese B encephalitis, and smallpox. By means of sanitary measures and specific immunization, Allied troops have been kept almost free of typhoid fever, typhus fever, and smallpox. Cases of dysentery, hepatitis, and encephalitis have been relatively few in number; current control measures are reasonably effective in preventing the importation of these diseases into the United States by returning troops, and research is directed toward improving preventive measures in order to eliminate even the few infections which do occur among men operating in the endemic area. Malaria, therefore, of these diseases, is one most likely to occur among veterans who have returned to the United States.

For the control of malaria, suppressive therapy with chloroquine is employed between April and October by all ground and air force personnel stationed in Korea. This is supplemented by mosquito control measures wherever possible, but the possibilities for individual exposure are greater in forward combat areas. Unavoidably, a certain number of men are bitten by infected mosquitoes, and the primary attack of malaria is usually suppressed by chloroquine therapy at that time. Since the predominant type of malarial parasite in Korea is a Temperate Zone variety of Plasmodium vivax which has a characteristically long latent period, the delayed primary attack may occur from 250 to 300 days after the infection, particularly if the individual was on suppressive therapy at the time of infection. Thus their attacks are usually seen in the late spring and early summer. For instance, among Army troops stationed in the United States, six malaria cases were reported in January of this year, eight cases in February, 24 in March, 74 in April, 309 in May, and 939 in June. Almost without exception, these patients contracted their infection in Korea. The Navy and Air Force have had comparable experiences, although of lesser magnitudes. A large number of cases may be expected in July with a diminution occurring in August and September, and another wave of attacks will probably occur next spring. Since it is neither desirable nor feasible to continue suppressive measures indefinitely and since there are no practical laboratory procedures for screening large numbers of men in order to determine infected individuals in advance of the primary attack, the Armed Services are investigating the possibility of using a new, relatively nontoxic, antimalarial compound for treating all men returning from Korea.

The treatment of cases which occur within the military service has constituted no particular problem. The cases have responded well to either chloroquine alone or chloroquine plus quinine. Although chloroquine does not prevent the occurrence of relapses of vivax infections, its administration is followed by a longer interval of freedom from attack than is observed with quinacrine (atabrine®) hydrochloride or quinine. It causes fewer reactions than other available drugs. The most frequent reaction is pruritus without rash, particularly of the palms, which occurs in about 10% of treated patients. Mild anorexia, nausea, and vertigo seldom justify discontinuance of the drug.

Chloroquine is available as chloroquine diphosphate, 0.5 gm. tablets (equivalent to 0.3 gm. base). The usual schedule used in military hospitals consists of two 0.5 gm. tablets initially followed by a single tablet after an interval of eight hours. Subsequently, single 0.5 gm. doses are given on each of the following two or three days. This schedule may be combined with quinine sulfate, 0.3 gm. orally three times a day, but ordinarily chloroquine alone is sufficient. In an endemic area, suppressive doses of chloroquine diphosphate, 0.5 gm., should be given weekly, beginning one week after the cessation of treatment and continued until four weeks after the last possible exposure.

If it were not for the fact that soldiers, sailors, and airmen occasionally have leave and many are now eligible for separation from military service, the delayed primary attacks and relapses of malaria that occur among men infected in Korea would all be treated in military hospitals. However, attacks do occur when individuals are absent from their military post, and as a result of the stepped-up separation program an increasing number will be experienced in civilian medical practice. It may be expected that the attacks will be diffusely scattered. Hence, members of the medical profession can expect to see some malaria cases among patients who are veterans of Korea.

In general, these cases of malaria are not expected to reestablish malarial infection in this country to any appreciable degree. Through cooperative efforts of the Federal, state, and local health authorities, and the practicing physicians, malaria has been almost eradicated in the United States. Control measures are now concentrated on the individual blood-positive cases. Thus, the success of the national program depends on early diagnosis and prompt reporting of all cases to appropriate health authorities. Human infection during the latent period generally is not communicable to mosquitoes. Patients with acute or chronic malaria are most infectious for mosquitoes. This period of communicability can be materially shortened by adequate medical treatment. Screening and residual insecticides should be utilized in the environment of each recognized case.

Army Postgraduate Program Extended.—A new series of postgraduate short courses for armed forces medical officers will considerably extend the Army's graduate professional education program, according to the Office of the Army Surgeon General. The new program is in keeping with the Army Career Management Plan, which provides a continuance of professional education throughout the military career of all Medical Corps and Dental Corps officers of the Army Medical Service.

A total of 18 courses will be given at five Army hospitals, the Armed Forces Institute of Pathology, and the Medical Service Graduate School, Washington, D. C., during the last six months of this year. Two courses of internal medicine are included. Other courses will be given in the second half of fiscal year 1952. (Naval and Air Force medical officers have been invited to attend these courses.)

The first of the two internal medicine courses, "Present Trends in Internal Medicine," will be given at Walter Reed Army Hospital, Washington, D. C., Oct. 8-12. Attendance will be limited to personnel from the First, Second, and Third Army Areas and the Military District of Washington. The second of the two courses, "Recent Advances in Internal Medicine," will be held at Letterman Army Hospital, San Francisco, Nov. 5-9. Attendance will be limited to personnel from the Fourth, Fifth, and Sixth Army Areas. Both courses will utilize lectures, conferences, round table discussions, and clinics.

Medical officers on active duty will be given priority in the allotment of spaces. Applications from medical officers on active duty will be forwarded through channels to: The Office of The Surgeon General, Department of the Army, Attention: Personnel Division, Washington 25, D. C. Applications from Air Force and Naval medical officers should be forwarded through regular channels. Applications from medical officers not on active duty and civilian physicians should be forwarded through channels to the commander of the installation offering the course. The commander will select those to attend on a space available basis. Army medical officers enrolled in the residency training program may attend these courses only if they are given at a hospital where the resident is being trained.

DEPARTMENT OF DEFENSE

August Induction of Medical Officers Delayed.—The planned induction under Selective Service of 333 Priority I medical officers scheduled during the month of August has been delayed temporarily at the request of the Department of Defense. The request was based entirely on the increased volunteer rate and does not represent a reduction in requirement for medical officers. Whether or not all of the doctors whose induction will be delayed during August will be called during September is dependent on the volunteer rate during the next few weeks. No reduction is anticipated for the call of 152 Priority I doctors scheduled to be inducted during September.

1. Released by the Office of the Surgeon General, Department of the Army, in cooperation with the Public Health Service; the Bureau of Medicine and Surgery, Department of the Navy; and the Office of the Surgeon General, Department of the Air Force.

FOREIGN LETTERS

SWEDEN

Blood Transfusion Service in Wartime.—At a meeting in April, 1951, devoted to a discussion of wartime problems, Dr. B. Vahlquist of Uppsala and Dr. O. Wilander of Ørebro dealt with the preparations for providing Sweden with a blood transfusion service capable of meeting wartime casualties. Some of their observations were based on experiences abroad. The French, for example, have calculated as the result of experiences in World War II that some 40 L. of blood must be provided for every hundred combatants. In 1949 there were some 30,000 blood donors available in Sweden, or 0.4% of the population. This is a much lower rate than that which existed in England in 1941, when there were some 900,000 donors available. Yet even the English did not have an overabundance of donors. What makes the situation worse, more than half the Swedish donors are at the present time men liable to be called into military service.

In recent months the Swedish Red Cross, in conjunction with the Swedish Army Medical Service, has conducted a campaign for recruiting donors in the county of Uppsala. Special attention has been paid to prospective donors among women between the ages of 18 and 50. Some 1,850 persons have been medically examined and tested for blood grouping. Among them were some 250 Rh-negative persons. Though this campaign was conducted with civil defense in view, results should prove of great value also in time of peace. The Swedish military authorities have organized the wartime blood transfusion centers so that about one-third of them are mobile, ready to take up work at new centers or to supplement stationary centers with personnel and equipment. It is realized that, in an emergency and when the blood supply is inadequate, it may be necessary to resort to substitutes, such as plasma and gelatin or saline solutions. It may also be necessary to relax some of the precautionary measures taken in peacetime. The suggestion that blood transfusion services should deal only with O-donors as universal donors has not found favor, as it would reduce the total number of donors by about 50%. Even in an emergency, particularly in wartime, one should not dispense with testing blood for syphilis.

Swedish Diabetes Association.—Ever since the successful application of the discovery of insulin, diabetics have increased in such numbers that effective cooperation among them has been called for with growing insistence. The local associations of diabetics established several years ago have combined in a central organization devoted to their interests. In 1943, an official investigation of diabetes in Sweden was started; in the autumn of 1948, this investigation culminated in a report in which a careful analysis of the diabetes situation formed the basis of certain definite plans for the future. In the autumn of 1949, the various diabetes associations were fused into the central Swedish Diabetes Association, charged with the task of research and treatment. Approximately half the members of the governing body of this association are doctors; among them were two, Prof. Hjalmar Holmgren and Dr. John Byttner, who recently died. These two deaths meant a serious loss to the association, for Prof. Holmgren was engaged on important research into disturbances of carbohydrate metabolism and the rhythmic activities of the liver with special reference to diabetes, and Dr. Byttner had taken an active part in the commission of inquiry between 1943 and 1948. He was engaged, at the time of his death, on plans for a special diabetes hospital, a center for research, teaching, and treatment.

These plans have now materialized in the conversion of a small private hospital into a center of research and treatment administered by the association. This center (Frejgatan 32, Stockholm) will begin with 16 beds for diabetics and will be in charge of Dr. Jakob Möllerström, who worked for many years with Prof. Holmgren. The clinical material available at this center will be closely studied with reference to such problems as those related to the so-called tertiary stage of diabetes, i. e., the changes to be observed in the eyes, kidneys, and vascular and nervous systems in diabetes of long standing. Doctors throughout Sweden have been invited to make use of and support this center.

Experiences of the Swedish Red Cross Hospital in Korea.—The Swedish Red Cross Hospital Unit which went to Korea in the fall of 1950, and which was stationed at Pusan, gained much valuable experience during the winter of 1950-1951. In a recent lecture in Stockholm about these experiences, Dr. G. Rundberg gave an account of the many precautions taken to combat all the evils of modern warfare in an uncivilized country. The members of the unit were inoculated against smallpox, typhus, typhoid, paratyphoid, cholera, tetanus, influenza, and, in a few cases, yellow fever. Inoculation with tetanus toxoid was so effective that no case of tetanus was observed among the United Nations' troops, whereas there were several such cases among the wounded prisoners of war admitted to the Swedish Hospital. The mortality was alarmingly high, despite treatment. Only one case of smallpox was admitted to hospital; it did not give rise to any secondary cases, presumably because of the immunity of the contacts. Up to February, 1951, there were 34 cases of typhus in a camp in which there were some 110,000 prisoners of war, but only in a few of these cases did the infection begin after admission to the camp. Though inoculation against typhus must not be under-rated, the most effective method of treating it seemed to be breaking the infection cycle by killing lice. Some seven tons of DDT were used as a spray every week.

Malaria caused by Plasmodium vivax was diagnosed in three cases in the hospital. Chloroquine phosphate appeared to be superior to quinacrine hydrochloride with regard to treatment and prophylaxis. As a prophylactic, chloroquine was given in tablet form only once a week. Many of the North Koreans treated in the hospital harbored ascarides. Dr. Rundberg stated that his American colleagues in a prisoner of war hospital had come to the conclusion that 90 to 100% of the North Koreans harbor these worms. The hospital was spared cases of rabies and plague, but the arrival of the latter was feared when the Chinese joined the North Koreans. During January and February, the venereal disease rate was high, with some 200 to 250 new cases daily. Prostitution being regarded as a legitimate occupation in Korea, and the number of prostitutes in Pusan alone being estimated at between 7,000 and 10,000, provision had to be made for care of the prostitutes as well as their clients

Death of Dr. Gunnar Nilson.—Dr. Gunnar Nilson was born on Sept. 28, 1872, in Uppsala, and died on March 28, 1951. He had for long been a striking figurehead in the Swedish medical world. The son of Prof. Lars Fredrik Nilson, he was educated in Uppsala and Stockholm, qualifying as a doctor in 1895 in the former town. He took his degree of doctor of medicine at the University of Lund in 1911, his subject being varicocele and its operative treatment. As early as 1898, he was attached to the Swedish Navy; in 1917, he was put at the head of the Swedish Naval Medical Service. He held this appointment till 1937, when he reached the age limit and retired. During the more than 20 years he was at the head of this service, Nilson's initiative and driving force were largely responsible for the reforms introduced. He was a clever and careful operator, and a good organizer and administrator. His personality was forceful, and he combined with it irresistible charm. With his patrician features and bearing, and because of the similarity of his name to that of England's great naval hero, he came to be known with affectionate jocularity by his colleagues as "The Sea Lord." He became a member of the Swedish Medical Society in 1900. From 1919 to 1940 he acted as secretary of the society as well as editor for its transactions. Much of his literary work was concerned with the history of medicine; in 1945, he published a volume on "Famous Swedish Doctors." In 1947, he published the "History of the Swedish Medical Society from 1908 to 1938." His latter years were disturbed by frequent small cerebral hemorrhages.

The items in these letters are contributed by regular correspondents in the various foreign countries.

SPAIN

Public Health Meeting.—The third Public Health Meeting was held in Madrid in April, 1951. The first and second meetings were held in 1943 and 1947, respectively. Dr. A. Crespo Alvarez and collaborators spoke on the use of antibiotics in sanitary campaigns, P. González and collaborators on modern trends in public health, and G. Clavero on virus diseases. Prof. G. Ramon (Paris) lectured on vaccination against diphtheria with diphtheritic antitoxin and vaccination against tetanus and associated vaccinations. Prof. Vogel (Hamburg) lectured on immunity in bilharziasis.

International Urology Forum.—The third international forum of urologic lectures was recently held at the urologic clinic of the Faculty of Medicine of the University of Madrid. Prof. A. de la Peña is the director of the clinic. Lectures were given by Dr. F. Orts Llorca, Faculty of Medicine of Cádiz; Drs. Sanz Ibañez, J. M. Corrales, Gay Prieto, and Vallejo Najera, Faculty of Medicine of Madrid; Dr. L. Caporale, Faculty of Medicine of Turin; Dr. D. Band, University of Edinburgh; Dr. C. de Moura, Faculty of Medicine of Lisbon, and Dr. V. Carulla, Faculty of Medicine of Barcelona.

For the first time since World War II, a group of internationally known professors of urology of faculties of medicine of Homburg-Sear, Diesseldorf, Erlanger, Koeln-Merheim, and Aschen have come to the Faculty of Medicine of Madrid, thus reestablishing cordial relations between German and Spanish urologists. They came to Spain on the invitation issued to the German Society of Urology. Dr. Boeminghaus, president of the German society, headed the group. Their lectures were as follows: Prof. Alken, renal vasography; Prof. Boeminghaus, trans-sacral cystectomy; Prof. Heuschi, chemotherapy of cancer of the urinary bladder; Prof. Tiermann, trans-sacral resection of hypogastric nerves, and Prof. Koch, lithogenesis and renal crisis.

Personals.—Dr. F. Grande Covián, having passed the examination, was recently appointed to the chair of physiology of the Faculty of Medicine of Zaragoza. Dr. Grande Covián collaborated with Prof. P. Hoffmann in Freiburg in 1929 on work on the physiology of the spinal reflexes. Later, he worked with Prof. S. Ochoa and Prof. J. Negrin in the Faculty of Medicine of Madrid on muscle chemistry. In 1932 and 1933, he worked in Copenhagen with Prof. Krogh on physiology of muscular exercise. In 1933, he worked in Lund with Prof. Thunberg on dehydrogenases of fatty acids. In 1934, he worked at the University College of London with Prof. L. Evans on metabolism of the heart. From 1936 to 1939, during the civil war in Spain, he was the assistant director of the Institute of Nutrition. He studied diseases of nutrition and the role of nicotinic acid in the prevention of pellagra. He found that pellagra may develop even in persons ingesting sufficient nicotinic acid. He proved that the neurologic symptoms are independent of other symptoms that appear in patients with pellagra and pointed out that the neurologic changes are not due to a deficiency in vitamin B_1. In 1941, he started a study on nutrition in Madrid for the Rockefeller Foundation, with emphasis on the effects of poor nutrition on physical development of children. From 1942 to 1944, he studied the vitamin content of Spanish food and the intermediary metabolism of carbohydrates. From 1944 to 1949, he conducted a large number of experiments on alloxan diabetes. In 1949, he was invited to work in the biochemistry laboratory of the Neuropsychiatric Research Center in Cardiff with Prof. Richter with a scholarship from the British Council for studies on the interchange of phosphorus in nerves and the use of chromatography in studying the metabolism of amino acids in the brain. Dr. F. Grande Covián is a member of the Physiological Society, the Biochemical Society, and the Nutrition Society of England, of the Danske Biologiske Selskabet of Copenhagen, and of the Sociedad Portuguesa de Biologia. He has been a constant collaborator of Prof. C. Jiménez Díaz, director of the Instituto de Investigaciones Médicas.

Dr. L. Cifuentes Delatte successfully completed an examination for the chair of urology in the Hospital de la Princesa in Madrid. Dr. Cifuentes Delatte was a disciple of his father, Prof. P. Cifuentes Díaz and of Prof. C. Jiménez Díaz of Madrid. In 1933, he worked in Berlin with Prof. A. V. Lichtenberg. In 1946, he studied transureteral resection in the United States under Prof. Nesbit of Michigan and Alcock and Flocks of Iowa. His article on epithelium of vaginal type in the female trigone was published in America. The existence of these islands of vaginal epithelium was confirmed by Hedberg of Sweden and by Vokaer of Belgium. He wrote a book on "Cystitis and Cystopathies."

MEXICO

Macrocytic Anemias.—During the last five years, the Hospital de Enfermedades de la Nutrición undertook the study and classification of macrocytic anemias, according to the observations made in patients from regions corresponding to the Mexican plateau (altitudes ranging between 1,600 and 2,200 meters above sea level). It was learned previously, in the same institution, that the mean corpuscular volume in normal subjects living in those areas is 80 to 105 cubic microns, which is 10 cubic microns more than values in the United States. This phenomenon was called physiological macrocytosis of the altitudes, as differentiated from the physiological polycythemia of altitudes, rarely found in Mexico.

The results obtained emphasize several facts:

1. Not a single case of pernicious anemia was found in Mexican patients, regardless of the length of the period of observation and despite the hospital's being an institution in which there is some concentration of hematological cases. Of two patients in whom the existence of pernicious anemia was suspected, one did not return to the hospital and in the other the final diagnosis was nutritional macrocytic anemia.

2. In a group of 70 patients with severe malnutrition, due exclusively to dietary deficiency, 40 had macrocytic anemia, which showed the clinical form of nutritional macrocytic anemia in 26 cases; in the other 14 the clinical picture was one of sprue. The criteria for considering one case as sprue, and not as nutritional macrocytic anemia, were based on the existence, besides the anemia, of severe diarrhea with steatorrhea, abdominal distention, flat curve of the oral glucose tolerance test, and hypocalcemia.

Achlorhydria was found in 55% of the sprue cases and in 50% of the nutritional macrocytic anemia series. Neurological alterations, which often were severe (subacute combined degeneration was observed several times), were characteristic of the nutritional macrocytic anemia patients, and absent in the sprue group.

The best therapeutic results in the macrocytic anemia patients were obtained with purified liver extracts and with vitamin B_{12}, which is at variance with the observations of other investigators.

3. In 27 cases of anemia of pregnancy, there was only one with macrocytic anemia.

4. Although anemia was frequent in a series of 111 patients with proved liver cirrhosis (an incidence of 62%), in only seven of them was it macrocytic; nutritional deficiency was accepted as the cause.

5. There were no cases of macrocytic anemia in which the disease was due to hypothyroidism or to Diphyllobothrium latum.

Poliomyelitis in Mexico City.—Until 1945, poliomyelitis was uncommon in Mexico. Since then, its incidence has been increasing; in 1950, nearly a thousand cases were observed in Mexico City (incidence in lesser cities and in the country has been lower), and the figures for the first half of 1951 seem to be higher. Several explanations have been offered for this epidemiological phenomenon, none of them absolutely satisfactory.

Poliomyelitis in Mexico City has certain peculiarities worth mentioning: 1. It occurs predominantly in children under 5 years of age. 2. The death rate has been low so far—no more than 5%. Nevertheless, in this year this proportion seems to have increased also. 3. Paralysis appears only in 15% of the cases and generally is not too severe or extensive. 4. Seasonal incidence, which parallels the warmer months, is strictly related to climatic conditions. In Mexico City, the outbreaks of the disease occurred in March or April. Federal authorities have adopted all necessary and known measures, and two weeks ago a committee with representatives of the health department, of the municipal government, and of the education ministry was created to study the implications and consequences of this disease.

CORRESPONDENCE

POLIOMYELITIS

To the Editor:—With reference to an article on "Second Attacks of Poliomyelitis" by Archibald L. Hoyne and Irving Lipton, published in THE JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION of June 23, 1951, I wish to call your attention to an article published by me on poliomyelitis in *California and Western Medicine*, June, 1946. This publication was the official publication of the California Medical Association on that date.

Drs. Hoyne and Lipton state in their summary, "It is probable that this is the only reported authentic second attack with fatal outcome." Perhaps the authors of this article did not completely review the literature, but I believe that the evidence presented in my article in *California and Western Medicine*, which is enclosed, leaves little doubt that this was also a second attack of poliomyelitis with fatal outcome. I am sure that this opinion was concurred in by Dr. Harold L. Faber, who was then chief of the division of pediatrics at Stanford Medical School. In the interest of scientific accuracy, I believe that THE JOURNAL might be interested in discovering that other second attacks that were fatal have occurred.

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HYSTERIA

To the Editor:—In the article by Drs. Purtell, Robins, and Cohen (*J.A.M.A.* **146:**902 [July 7] 1951) the point is well made that "hysteria" can be suspected clinically with as much accuracy as "organic" disease. However, I feel that the name used, "hysteria," is archaic and had best be abandoned, for the following reasons: 1. It has no precise definition in either medical or lay usage. 2. It is not a disease entity, nor can it be called a syndrome. 3. Its derivation (uterus) is misleading. 4. Other terms in current usage give a better picture of the mechanisms involved.

Psychiatric literature suffers from a confusion and profusion of terms; by its very antiquity, "hysteria" is a prime example of this.

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MORE ABOUT CLOTTING IN BIBLICAL LITERATURE

To the Editor:—Dr. Cyril E. Bousfield's comment on page 1152 of THE JOURNAL for July 21 requires further comment. Where is the evidence that Jesus was dead when speared? His companions' legs were broken—a custom based on experience to keep the crucified from reviving and walking away. A spear was thrust into His side to make sure He was dead. The fact that blood came when the skin was punctured makes it likely that He was alive. The "water" is another thing entirely. Even a small amount of blood mixed with it at any time prior to the deep puncture wound would have given the appearance of blood; hence the word water would not have been used. One must conclude that an effusion was tapped, either pericardial or pleural.

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INFORMATION ON BITES FROM REPTILES AND INSECTS

To the Editor:—I have been endeavoring to obtain statistical data on the types of poisonous reptiles and insects in the various states together with the number of persons bitten each year and the number of deaths. I am having somewhat of a difficult time because practically all states do not require reports of bites or stings of poisonous reptiles or insects. State boards of health have been very kind in replying to a questionnaire giving such meager information as they have.

I have come to the conclusion that the only way I can really get much information on the subject is by contact with medical men. Since it is impossible to write each doctor and to bear the expense of correspondence, it has seemed to me that THE JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION might serve as a medium for obtaining that information. The statistical data required are as follows: 1. Number of persons treated in 1950 for poisonous reptile and insect bites (list kind of reptile or insect). 2. Number of persons treated in 1949 for poisonous reptile and insect bites (list kind of reptile or insect). 3. Number of deaths.

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MEDICAL MOTION PICTURES

The Stowaway: 16 mm., black and white, sound. Part I, Disease and Personal Hygiene (TF 8-1501), 17 min. Part II, Galley Sanitation (TF 8-1502), 24 min. Part III, Food Storage (TF 8-1503), 12 min. Produced in 1949 by the U. S. Army. Procurable on loan from Army Surgeon of Army Area in which the request originates, or on purchase from United World Films, Inc., 1445 Park Ave., New York 29.

Part I, Disease and Personal Hygiene, reveals both obvious and obscure ways in which disease is spread by food-handling personnel and clearly illustrates proper sanitary precautions and remedies. The methods shown in this film will help eliminate the spread of disease caused by careless personal hygiene of food handlers.

Part II, Galley Sanitation, presents methods of sanitation observed in a ship's galley as an illustration of how food service organizations should exercise every precaution to insure all sanitary measures in preventing the spread of disease.

Part III, Food Storage, covers the careful inspection of food by the U. S. Army Veterinary Corps, the Bureau of Animal Industry and the Port Food Service Inspector prior to procurement and use aboard troop transports. It shows in detail the proper facilities for food storage and the principles used for the proper storage and preservation of food.

Part I approaches the problems of causes of contamination negatively, showing how all the rules of hygiene can be and are broken. Parts II and III are "how to do it" films in which the teaching is accomplished by positive methods. They provide motivation by the use of excellent, clearly illustrated examples of why and how to store food properly. Repetition is used very effectively as a teaching method. Although these films are primarily concerned with the training of food handlers on troop transport ships, the lessons taught are applicable to other situations. These films are highly recommended for the beginning and in-service training of all personnel engaged in or planning to engage in food handling; however, certain aspects of Part I limit its use to male audiences. The photography and sound are excellent.

Surgical Anatomy of the Inguinal Area: 16 mm., color, silent, showing time 29 min. Prepared by Conrad J. Baumgartner, M.D., and De Vere W. McGuffin, M.D. Produced in 1947 by Department of Visual Education, College of Medical Evangelists, 312 N. Boyle Ave., Los Angeles 33. Procurable on loan from Davis & Geck, Inc., 57 Willoughby St., Brooklyn 1.

This film shows the anatomy encountered in the repair of an inguinal hernia with particular attention to the transversalis fascia and Cooper's ligament. The dissection is performed on a fresh cadaver. The anatomy of the inguinal region is unusually well presented. The relation of the transversalis fascia to the femoral sheath and the location of Cooper's ligament are beautifully demonstrated. The anatomic structures used in the repair of the inguinal hernia are much better shown than on most films on the technic of inguinal hernia repair. The film is well captioned. This is an excellent teaching film and could be recommended for use in the teaching of anatomy to first year medical students as well as for the teaching of the technic of hernia repair to third and fourth year medical students. It would also be applicable for the teaching of interns, residents, and surgeons.

BUREAU OF LEGAL MEDICINE AND LEGISLATION

MEDICOLEGAL ABSTRACTS

Prenatal Injury: Liability for Stillbirth Following Motor Bus Accident.—The plaintiff and her husband filed suit for damages alleged to have resulted from injuries sustained by the plaintiff in a motor bus accident. From a judgment for the plaintiff, the defendant appealed to the court of appeal of Louisiana.

The motor bus, which was owned and operated by the defendant corporation, was driven into a ditch and the driver attempted to extricate it before allowing the passengers to alight. Although, in certain parts of the record, it is contended that the plaintiff was thrown to the floor of the bus, this did not occur; she caught herself on the seat ahead of her and received, as a matter of fact, only a slight jolt. At the time of the accident, however, the plaintiff was two to four months pregnant and about five and one-half months later the child with which she had been pregnant was born dead.

The major item of loss claimed by the plaintiff was based on the fact that the expected child was born dead, and the defendants contended that for such an item of loss or damage there can be no recovery, as a matter of law, regardless of the cause.

The defendant directed the court's attention to Article 28 of the Civil Code providing: "Children born dead are considered as if they had never been born or conceived." The defendants argued that since a child born dead is considered as never having been conceived, it must be concluded that nothing has been lost since there can be no loss of anything that has never existed. We can not be persuaded, said the court, that there are no circumstances under which damages should be awarded to the parents of the unborn child if the foetus, while in its mother's womb, has been so injured that it cannot be borne alive. We are certain that the redactors of our code had no such purpose in mind in enacting Article 28.

The court was referred to a discussion on the subject of "Tort Liability for Prenatal Injury" in 24 T. L. R. at page 435. The author of that article says that as a general rule "there can be no recovery for the loss of the society and prospective earnings of the child." There could, however, be a recovery for a result of an accident which might cause sterility, or which might otherwise prevent parents from having children, said the court. If, as the result of actionable negligence, a husband or wife should be so injured that either, in the future, could not expect to produce children, surely this would be taken into consideration as an item of damage. It necessarily follows that when parents are actually expecting arrival of a child, and are deprived of the fruition of that great expectation by someone's actionable negligence, they may recover from the tortfeasor for that particular loss. In the article above referred to, the author also says "although mental anguish based specifically upon the loss or death of the child is not recoverable, recovery has been allowed for the mother's mental anguish if the child is born malformed." If the mother may recover for the mental anguish caused by the fact that the child has been born malformed, said the court, it is surely illogical to hold that, under the same circumstances, the mother cannot recover where, as the result of negligence, the child is prevented from coming into existence at all. In the same article the author states that, where there is an injury to a pregnant woman "there may be recovery for the fear that harm may have resulted to the child." If this is true, surely it should follow that if that fear is realized and the child is stillborn, there should be allowed recovery for the stillbirth of the child. As a matter of fact, said the court, the author of the article concludes with a statement that, in all such cases in which it is contended that some accident has caused prenatal injuries, "the evidence to prove causal relationship should be subjected to the strictest examination." And that is our conclusion here, said the court of appeals, that as a matter of law, there may be recovery by the parents where there is actionable negligence which causes such injury to a viable foetus as to prevent its being born alive, but that there must be strict proof that the actionable negligence was the cause of the unfortunate occurrence.

The court next considered whether or not the plaintiffs introduced sufficient evidence to indicate that the stillbirth was caused by the accident. The plaintiff contended that the accident made it necessary for the plaintiff to remain in bed for a large part of the time, that she remained inactive and took no exercise, and that as a result her weight became very excessive, with the secondary result that the weight of the viable foetus became also excessive and that, as a consequence, the foetus itself grew so large that it could not be born alive. Dr. LaRocca, the attending physician, did not attribute the stillbirth directly to the accident. He said: "I did not say the trauma caused the death. I said that the oversize of the baby, and the abnormal circumstances of her pregnancy, were the cause of the death of the baby." He explained that what he meant by "abnormal" condition of her pregnancy, was the fact that the plaintiff was confined to her bed for long periods, with the result that she and the baby grew to abnormal proportions. We are of the opinion, said the court, that the overwhelming weight of medical testimony is to the effect that these factors had nothing to do with the somewhat abnormal development of the foetus. The testimony of a number of doctors indicates that no matter how much an expectant mother may eat, the foetus absorbs only so much as nature requires, and that its size does not depend at all on the quantity of food taken by the mother. These doctors also stated that the fact that the mother is required to remain in bed for several hours each day and that she is permitted to take little or no exercise does not affect the size of the foetus.

We conclude, as we have stated, that the abnormal size of the foetus was not the result of the fact that the mother herself was tremendously overweight and that she was required to remain inactive for several hours each day. In reaching this conclusion, the court continued, we have not been able to lose sight of the fact that the record strongly suggests another cause for the abnormal size of the unborn foetus. All of the doctors agree that diabetes of a mother is almost always the cause of abnormal growth of the foetus. While Dr. LaRocca stated that the plaintiff did not have true diabetes, he conceded that she had what he said was "transitory" diabetes, and the diet which he required for her was one which is recognized for diabetes. He says, too, that he took into consideration the fact that the plaintiff was much overweight and that she was confined to her bed for long periods and prescribed a diet accordingly, and he very significantly added, "If she had lived up to it her weight would have been reduced." The medical experts who testified on behalf of the defendants stated that they did not know of such disease as transitory diabetes but there can be no doubt that the overwhelming medical testimony shows conclusively that the percentage of abortions and stillbirths among diabetic women is tremendously greater than among women not so affected.

Even if it could be conceded that the size of the baby was not the result of the mother's tendency to diabetes and was not the result of the extreme overweight of the mother or of her long periods of rest, still there is no proof whatever that its size was the cause of its being stillborn. Dr. LaRocca knew of its size. He had made all necessary examinations of the mother and at the time he knew of nothing which would have prevented her from giving normal birth to a child even of that size. We cannot say how much significance should be attached to it, but we have not been able to lose sight of the fact that although the plaintiff had given birth to her first child, which was still living, she had, some five years before the present occurrence, given birth to another stillborn child, thus possibly indicating a tendency which may have been felt at this time.

Our conclusion is that at most all that can be said is that there was a sequence of events which may have led a layman to feel that the stillbirth was the result of the accident, but that the overwhelming medical testimony makes it impossible that we accept that conclusion and we have decided that no allowance should be made for the fact that the child was not born alive.

The court therefore held that the parents had a right to maintain an action for damages for the stillbirth but that they failed to prove that the stillbirth was caused by the accident. Other items of damages were, however, awarded, and the judgment in favor of the plaintiff was affirmed.—*Valence v. Louisiana Power & Light Co., 50 So. (2d) 847 (Louisiana, 1951).*

MEDICAL LITERATURE ABSTRACTS

AMERICAN

A.M.A. Arch. Indust. Hyg. & Occup. Med., Chicago

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Aiding Cardiac Patient in Industry. E. M. Kline.—p. 454.
Air Flow Measurements on Human Subjects With and Without Respiratory Resistance at Several Work Rates. L. Silverman, G. Lee, T. Plotkin and others.—p. 461.
Chronic Toxicity of Methylpolysiloxane "DC Antifoam® A" In Dogs. G. P. Child, H. O. Paquin Jr. and W. B. Deichmann.—p. 479.
Activities of State and Local Agencies in Industrial Health. V. M. Trasko.—p. 483.
Cadmium Poisoning. L. H. Cotter and B. H. Cotter.—p. 495.
Investigation of Small, Airborne, Charged Particles by Electrical Method. J. H. Daniel and F. S. Brackett.—p. 505.

3:543-648 (June) 1951

SYMPOSIUM ON TREATMENT OF CHRONIC BERYLLIUM POISONING WITH ACTH AND CORTISONE

Chronic Pulmonary Interstitial Granulomatosis: Preliminary Report on Two Patients Treated with ACTH. J. M. De Nardi.—p. 543.
Case of Chronic Beryllium Poisoning from Atomic Energy Development. R. J. Hasterlik.—p. 547.
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*Spontaneous Remission of Chronic Berryllium Posioning from Fluorescent Lamp Manufacturing: Report of Case. J. W. Cass Jr.—p. 569.
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Beryllium Disease from the Ceramic Industry: Report of Case. P. W. Reynolds.—p. 575.
Metabolic Study of Case of Chronic Beryllium Poisoning Treated with ACTH. H. L. Hardy, F. C. Bartter and A. E. Jaffin.—p. 579.
Choice of Drugs and Dosage. H. S. Van Ordstrand.—p. 583.
Recognition and Prevention of Complications of ACTH and Cortisone Therapy. T. F. Frawley.—p. 587.
Information Gained in Pretherapy and Post-Therapy Pulmonary Function Studies. J. McClement.—p. 599.
Pulmonary Function in Patients with Pulmonary Disease Treated with ACTH. B. G. Ferris Jr., J. E. Affeldt, H. A. Kriete and J. L. Whittenberger.—p. 603.
Interpretation of Results of ACTH and Cortisone Therapy in Chronic Beryllium Poisoning: Data Obtained by Pretherapy and Post-Therapy Studies of Pulmonary Function. G. W. Wright.—p. 617.
Minimum Observations Necessary to Advance Knowledge of Action of ACTH and Cortisone. A. Forbes.—p. 622.
Current Research Problems Concerning Pulmonary Granulomatosis in Beryllium Workers. F. Klemperer.—p. 625.

Spontaneous Remission of Chronic Beryllium Poisoning.—Cass describes the case of a man who, in January, 1943, complained of shortness of breath, loss of weight, and general weakness. Roentgenograms of the chest led to a questionable diagnosis of sarcoid, but further inquiry revealed that in 1941 the man had worked in a fluorescent lamp factory. Later it was recognized that this man had a syndrome similar to that found in several other employees of this company and that it was probably beryllium poisoning. The patient remained disabled and acutely ill for six or seven months. Finally he began to improve. At the end of eight months he was well enough to go back to part-time work, and about one year after the onset of the illness he was able to return to full-time work. Roentgenograms of the chest still show diffuse granulomatous lesions, but the individual nodules seem smaller and more dense than formerly. Small stones were passed during several episodes of acute renal colic, and their analysis revealed beryllium, but the last time stones were passed, they showed no evidence of beryllium. The patient received medical supervision but no specific therapy. The author doubts that either corticotrophin (ACTH) or cortisone should be used for this disease over long periods as has been suggested by some. He feels that as adjuncts to other symptomatic therapy during the phase of prostration and debility, as a means of producing general systemic stimulation, their use may be justified, but there is little justification for using them simply because the roentgenograms show improvement of the pulmonary lesions. In fact, this may be a disadvantage, for the removal of the protective band of lymphocytes from the beryllium particles may result in an undesired freeing of beryllium.

A.M.A. Arch. Otolaryngology, Chicago

53:483-606 (May) 1951

Distribution and Comparison of Nasal Cilia. G. E. Tremble.—p. 483.
Uncommon Conditions in Field of Otolaryngology Seen in Office Practice. J. C. Peele.—p. 492.
*Is Hearing Loss Due to Nutritional Deficiency? Further Studies on Influence of Vitamin A in Certain Types of Impaired Hearing. M. J. Lobel.—p. 515.
Secondary Tonsillar and Adenoid Hemorrhage in Relation to Barometric Pressure Changes. S. J. Pearlman and A. Loewy.—p. 527.
Treatment of Complete Unilateral Bony Atresia of Posterior Nares: New Technique and Brief Reference to Asphyxia Neonatorum. H. H. Beinfield.—p. 530.
Allergy: Summaries of Bibliographic Material Available in Field of Otolaryngology for 1949. E. L. MacQuiddy and E. A. Holyoke. —p. 560.

Hearing Loss and Nutritional Deficiency.—Lobel cites several investigations on the value of an injectable vitamin A preparation in otosclerosis and other types of impaired hearing not amenable to the usual otolaryngologic methods of treatment, and then describes patients with hearing loss who have been treated primarily with vitamin A and other measures. He concludes that the present clinical study, like some previous ones, indicates that vitamin deficiency and hearing loss are in some manner related. Results of clinical trials with an injectable vitamin A preparation hold promise of its therapeutic value in certain types of deafness. Despite the fact that in some instances the hearing loss was not influenced, the accompanying tinnitus was greatly relieved. Further trials with vitamin A, alone or combined with other vitamins, seem definitely warranted.

American Journal of Hygiene, Baltimore

53:237-358 (May) 1951. **Partial Index**

In Vitro Sensitivity Tests with Eight Antibiotics Against Escherichia Coli 111, B4, Special Type of Coliform Bacillus Associated with Infant Diarrhea. W. W. Ferguson, J. C. Jennings and R. Y. Gottshall. —p. 237.
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Japanese B Encephalitis Virus in Blood of Experimentally Inoculated Birds: Epidemiological Implications. W. M. Hammon, W. C. Reeves and G. E. Sather.—p. 249.
Study of Association of Factors of Pregnancy and Parturition with Development of Cerebral Palsy: Preliminary Report. A. M. Lilienfeld and E. Parkhurst.—p. 262.
Antigenicity of Single, Graded Doses of Purified Diphtheria Toxoid in Man. G. Edsall, H. J. Banton and R. E. Wheeler.—p. 283.
Immunological Relationships Among Species and Strains of Virulent Treponemes as Determined with Treponemal Immobilization Test. A. S. Khan, R. A. Nelson Jr. and T. B. Turner.—p. 296.
Immunization Against Scrub Typhus: I. Combined Living Vaccine and Chemoprophylaxis in Volunteers. J. E. Smadel, H. L. Ley Jr., F. H. Diercks and others.—p. 317.
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Role of Particle Size in Experimental Air-Borne Infection. L. S. Sonkin. —p. 337.

The Association library lends periodicals to members of the Association and to individual subscribers in Continental United States and Canada for a period of five days. Three journals may be borrowed at a time. Periodicals are available from 1940 to date. Requests for issues of earlier date cannot be filled. Requests should be accompanied with stamps to cover postage (6 cents if one and 18 cents if three periodicals are requested). Periodicals published by the American Medical Association are not available for lending but can be supplied on purchase order. Reprints as a rule are the property of authors and can be obtained for permanent possession only from them.

Titles marked with an asterisk (*) are abstracted.

American Journal of Medical Sciences, Philadelphia

221:479-598 (May) 1951

*Prevention of Degenerative Vascular Lesions in Young Patients by Control of Diabetes. J. L. Wilson, H. F. Root and A. Marble.—p. 479.
Incidence of Diabetic Retinopathy Relative to Degree of Diabetic Control. S. Spoont, W. W. Dyer, R. Day and H. Blazer.—p. 490.
Iatrogenic Hypothyroidism. F. T. Billings.—p. 495.
*Effect of Cortisone on Clinical Course of Chronic Regional Enteritis and Chronic Idiopathic Ulcerative Colitis. T. E. Machella and O. R. Hollan.—p. 501.
Myocardial Infarction: Study of Acute Phase in 920 Patients. F. J. Smith, J. W. Keyes and R. M. Denham.—p. 508.
Bronchiectasis: Study of 220 Proved Cases. T. H. Lorenz.—p. 522.
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Satiety as Factor in Nutrition States: Observations on Mass Exchange. J. M. Strang.—p. 537.
Whipple's Intestinal Lipodystrophy: Its Relationship to Rheumatic State. J. C. Peterson and R. H. Kampmeier.—p. 543.
Evaluation of Artane and Tolserol in Treatment of Spastic Disorders. A. S. Effron and W. M. Schultz.—p. 561.
Recent Advances in Drug Therapy of Peptic Ulcer. J. E. Berk.—p. 567.
Roentgenologic Diagnosis of Congenital Cardiovascular Malformations. D. M. Gould.—p. 581.

Prevention of Degenerative Vascular Lesions in Diabetes Mellitus.—Studies were made of 247 patients with diabetes mellitus, 122 males and 125 females, with age of onset from 18 months to 30 yr. and with duration from 10 to 34 yr. Particular emphasis was given to the relationship between control of the diabetes mellitus and the development of degenerative lesions in the kidneys, retinas, and blood vessels. Ideal or perfect control of diabetes mellitus implies that a balanced diet has been so well utilized by an adequate insulin effect that blood and urine tests are invariably normal. When various degrees of control were plotted against the duration of diabetes and the development of calcified vessels, retinitis, and albuminuria, the following facts appeared: No patients with excellent or good control showed advanced calcification or pronounced retinitis even after periods of 20 to 34 yr. of diabetes mellitus. Diabetic nephropathy did not develop in patients with good or excellent control, even after periods of 20 to 34 yr., but did appear in 62 patients with poor or fair control, 13 of whom died during the year of this study. Four out of 103 patients with diabetes for periods of at least 20 yr. had excellent control. Three of these had no retinal lesions. Of 54 with moderate to pronounced retinitis, all but five had had fair or poor control and none had had excellent control. Of 80 patients who lived for periods of at least 20 yr. on an unmeasured diet without constant use of insulin and without constant control of glycosuria, 55 showed severe degenerative vascular lesions. Whatever the specific etiological factors causing diabetic degenerative lesions may be, it has been demonstrated that the regulation of diabetes mellitus controls these factors. Control of diabetes mellitus is thus more important than any known factor, such as duration or severity, in prevention of these degenerative complications.

Cortisone in Chronic Regional Enteritis and Chronic Ulcerative Colitis.—Three patients with chronic regional enteritis with or without involvement of the proximal colon and three patients with chronic ulcerative colitis were subjected to a therapeutic trial with cortisone. The drug was injected subcutaneously according to the following schedule: 100 mg. every eight hours for three doses, then 200 mg. every 12 hr. for two doses, and thereafter 50 mg. every 12 hr. for eight additional days. Moderate to pronounced remission of symptoms occurred in the three patients with regional enteritis and in two of the patients with ulcerative colitis. In the three patients with regional enteritis the improvement seemed to be definitely related to the cortisone; the evidence was less clear in the two patients with ulcerative colitis. In each of the five improved patients a relapse occurred within five to 73 days after cessation of treatment. The best responses were obtained in the two patients who had the lowest initial eosinophil counts. The patient with the highest eosinophil count showed no improvement. Two of the three patients who responded most dramatically were the most critically ill of the group, which suggests that the severity of the disease is one of the factors determining a good response. A trial of cortisone therapy would appear to be worth while in certain cases of regional enteritis or ulcerative colitis, particularly in critically ill patients when the situation is desperate and a long enough period of improvement is desired for the effective use of other therapeutic measures. Cortisone does not ensure cure of the disease, as a relapse may occur after administration of the drug is discontinued.

American Journal of Physiology, Baltimore

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Decrease of Cardiac Output by Acute Pericardial Effusion and Its Effect on Renal Hemodynamics and Electrolyte Excretion. R. S. Post.—p. 278.
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American Journal of Psychotherapy, New York

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Am. J. Roentgenol. & Rad. Therapy, Springfield, Ill.

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*End Results of Radiation Treatment of Cancer of Cervix Uteri. H. E. Schmitz.—p. 715.
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Individualized Interstitial Irradiation of Cancer of Uterine Cervix Using Cobalt 60 in Needles, Inserted Through Lucite Template: Progress Report. J. L. Morton, A. C. Barnes, G. W. Callendine Jr. and W. G. Myers.—p. 737.
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Dose Distribution Measurements with University of Illinois 25 Mev. Medical Betatron. J. S. Laughlin, J. W. Beattie, J. E. Lindsay and R. A. Harvey.—p. 787.
Practical Compression Device for Spot Roentgenograms. W. S. Tirman and A. C. Nickel.—p. 800.

Growth and Spread of Cancer of Cervix Uteri.—Cancer of the cervix uteri may begin as carcinoma in situ, a disease that has been detected in patients whose average age is eight to 12 years younger than that of women with infiltrative cancer of the cervix. If it can be shown that carcinoma in situ is the usual precursor

of infiltrative cancer, these figures would suggest that there may be a prolonged period during which incipient cancer may be detected and treated with the prospect of close to 100% cure rate. On the contrary, untreated invasive cancer of the cervix is a rapidly fatal disease. Of 171 women with cancer of the cervix seen at the New York Cancer Institute between 1930 and 1949 who had not received surgical or irradiation treatment, 58% died within one year of the onset of symptoms and 82% within two years. Four and six-tenths per cent of the women with untreated cervical cancer survived five years. Radical hysterectomy was performed on 91 women with cancer of the cervix. Microscopic examination of the parametrial tissue outside the cervix showed invasion by cancer in about half the patients, proving that parametrial involvement occurs by direct extension fairly rapidly. Only 15 (16.5%) of these 91 patients showed lymph node metastases. The nodes in the obturator fossa were the sites of metastasis in nine patients, the lymph node lying in the bifurcation of the common iliac artery ("hypogastric node") in eight patients and in the common iliac nodes in four patients. Necropsy in patients who died of cancer of the cervix revealed metastatic disease outside the pelvis in only about 50%. The commonest sites of metastasis were the periaortic and retroperitoneal nodes, the liver, the lung, and the vertebral column. The commonest cause of death in cancer of cervix uteri is either ureteral obstruction or infection of the urinary tract associated with it. Operative relief of such obstruction is of doubtful value, unless there is some promising method of curing the primary tumor.

Irradiation Treatment of Cancer of the Cervix Uteri.—A questionnaire sent to the heads of the departments of gynecology of the 77 medical schools in the United States and Canada demonstrated that these physicians prefer irradiation treatment to surgical treatment of cervical cancer. This report shows the end results of a planned course of irradiation therapy in 188 patients with cervical carcinoma who had no previous treatment. They were given 50 mg. of radium element in tandem filtered by 2 mm. of brass and 3 mm. of para rubber for 1,500 mg. hr. every eight days in three applications for a total of 4,500 mg. hr. The radium dose administered to area A was 7,000 gamma roentgens and, to area B, 1,540 gamma roentgens. On the days the patient was not receiving radium therapy, she was given daily roentgen treatment to one field; 250 or 300 r was administered daily, and the total number of fields was regulated by the measurement of the patient. A total tumor dose of 3,500 to 4,000 r was given. Of the 188 patients, 43.6% are free of evidence of disease after five years. The complications associated with irradiation of the pelvis were not found to be frequent or severe enough to cause rejection of this method of therapy. The cell type of the disease in no way aided in prognostication. The response of the tumor to therapy and the extent of the disease definitely influenced the prognosis. It is therefore recognized that any pronounced progress in saving the life of patients with carcinoma of the cervix must be made through early detection of the lesion. There is no evidence that primary surgical treatment holds any advantage over irradiation therapy.

American Journal of Surgery, New York

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Brachial Plexus Anesthesia: Comparative Study of Agents and Technics. W. M. Shaw and B. Root.—p. 407.
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*Thrombo-Embolism in Children: Analysis of 35 Cases. P. T. Decamp, A. Ochsner and M. DeBakey.—p. 611.
Cardiac Tamponade: Treatment by Aspiration. D. C. Elkin and R. E. Campbell.—p. 623.
Acute Appendicitis in Aging Negro. F. F. Boyce.—p. 631.
Total Intravenous Alimentation in Treatment of Small Bowel Fistulas. H. C. Hull and T. G. Barnes.—p. 644.
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*Indications for Surgical Intervention in Ulcerative Colitis. F. H. Lahey.—p. 726.
One-Year to Four-Year Follow-Up Examinations of 130 Vagotomized Patients. W. Walters and H. H. Belding III.—p. 743.

Thromboembolism in Children.—During the 12-yr. period ending June 30, 1950, thromboembolism occurred in 35 children aged 12 or less who were admitted to a large general hospital. While the over-all incidence of thromboembolism is lower in children than in adults, a higher percentage of the cases in children are fatal. The age distribution of the fatal cases in children closely followed the age distribution of all fatalities in the hospital, the highest incidence occurring in early infancy. In contrast with thrombosis in adults, major precipitating factors are essential for thrombosis to develop in children. The newborn infant is particularly susceptible to such precipitating factors as infection, dehydration, malnutrition, and circulatory stasis. Nonseptic peripheral thrombosis was rarely observed in the children reported on. Visceral thrombosis was common and frequently contributed directly to a fatal outcome. Pulmonary embolism, thrombosis, or both, were frequently observed in younger and older children. Heart disease was a significant cause of thrombosis in older children. The incidence of thrombosis in children has diminished during the period of the author's study, while the adult incidence has doubled. This improvement resulted primarily because of the control of infections previously leading to venous thrombosis in children. The incidence should be further reduced as the principles and techniques of hydration, nutrition, and control of regional and systemic infections are applied more vigorously.

Surgical Intervention in Ulcerative Colitis.—Of 770 patients with ulcerative colitis treated at the Lahey Clinic, 308 required surgical therapy. Ileostomy was the procedure most frequently performed. Because of improvement in the medical and surgical management of these patients the mortality rate for this operation has been reduced from 18.6% to 2% since 1947. An important medical advance has been the use of corticotrophin (ACTH), which brought about a remission in 21 out of 28 patients in this series. This made it unnecessary to perform ileostomy and colectomy during the acute stage, when the mortality rate is high. Early ileostomy is justified in a patient in the acute stage of the disease when remission has not occurred promptly with corticotrophin. The performance of ileostomy and its management has been improved, so that complications such as skin erosion, prolapse, and retraction of ileostomy no longer occur. Bowel hemorrhage in the acute phase of the disease is another indication for prompt ileostomy. If colectomy then becomes urgently necessary because of failure to control the bleeding from the colon, it can be undertaken with the ileostomy already established and the fecal stream sidetracked. Ileostomy and partial resection of the colon combined in one operation may elevate the mortality rate unduly and should be done only in good risk patients by surgeons with experience in these cases, and even then with extreme caution. Acute perforation has not been a common complication in the author's experience. Chronic perforation was frequently associated with obstruction and with a walled-off abscess. All closures of perforations for ulcerative colitis should be combined with ileostomy. Involvement of the joints is a definite indication for prompt ileostomy, to be followed by total colectomy and abdominosacral removal of the rectum. The same applies to multiple anal fistulas that produce induration of the perineal tissues and resultant rigid anal sphincters. Colectomy is advised for all patients who have had a permanent ileostomy established.

Arkansas Medical Society Journal, Fort Smith

47:223-242 (May) 1951

Use of Anticoagulants in Diseases of Circulation. C. T. Chamberlain. —p. 223.
Breast Cancer. Brief Review of Theory and Therapeutics. H. G. Hollenberg, W. G. Cooper Jr. and R. D. Jones.—p. 226.
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Cancer Research, Chicago

11:297-392 (May) 1951. **Partial Index**

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Increased Weight and Mitotic Activity in Liver of Tumor-Bearing Rats and Mice. E. Annau, A. Manginelli and A. Roth.—p. 304.
Distribution of S^{35} from Labeled DL-Methionine in Mice Bearing Carcinoma of Breast, Neoplasms of Hematopoietic System, or Liver Abscesses. H. S. Bloch, C. R. Hitchcock and A. J. Kremen.—p. 313.
Carcinogenic Hydrocarbons and Related Compounds in Processed Rubber. H. L. Falk, P. E. Steiner, S. Goldfein and others.—p. 318.
Carbohydrate Metabolism of Brown-Pearce Carcinoma of Rabbit in Normal and Hypoglycemic Serum. R. H. Steckel and J. R. Murlin. —p. 330.
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Systemic Effects of Tumors in Rats. R. W. Begg.—p. 341.
Histochemical and Microchemical Changes in Experimental Cirrhosis and Hepatoma Formation in Mice by Carbon Tetrachloride. R. E. Stowell, C. S. Lee, K. K. Tsuboi and A. Willasana.—p. 345.
Histological and Chemical Alterations Produced in Mouse Epidermis by Soft X-Radiation. M. H. Toosy.—p. 361.
Studies on Tumor-Retarding Agent Produced by Aspergillus Fumigatus. H. C. Reilly and C. C. Stock.—p. 366.
Fluorescence Studies of Carcinogens in Rat Skin. P. Cambel.—p. 370.
Electrophoretic and Ultracentrifugal Studies on Soluble Proteins of Various Tumors and of Livers from Rats Fed 4-Dimethylaminoazobenzene. S. Sorof and P. P. Cohen.—p. 376.
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Diseases of Chest, Chicago

19:493-614 (May) 1951

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Angiocardiographic Findings in Pulmonary Tuberculosis. I. Steinberg, H. I. McCoy and C. T. Dotter.—p. 510.
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Experiences with Dry BCG Vaccine in the Philippines. S. Bona-de Santos, F. Duñgo and A. Aoanan.—p. 544.
*Role of Para-Aminosalicylic Acid (PAS) in Emergence of Streptomycin-Resistant Tubercle Bacilli. W. G. Lewis, L. S. Arany and B. H Johnson.—p. 566.
Psychogenic Factors in Chest Diseases. A. S. Anderson.—p. 570.
Classification of Pulmonary Tuberculosis with Five Digit Code. B. Papanicolaou.—p. 577.
Standardized Oblique Roentgenography of Chest. S. W. Atwell.—p. 585.
Benign Fibroma of Mediastinum. J. W. Fouche.—p. 589.
Migratory Pneumonia Without Eosinophilia. D. Biber and A. Buckler. —p. 593.
Spontaneous Rupture of Esophagus. H. L. Rigdon and N. D. Ellis. —p. 598.

PAS and Resistance of Tubercle Bacilli to Streptomycin.—The studies reviewed in this paper were carried out in participation with the Veterans Administration-Army-Navy Research Program, which used paraaminosalicylic acid in combination with streptomycin. The aim was to delay streptomycin resistance by lowering of the total dosage of streptomycin and by addition of a synergistic drug. Streptomycin resistance was studied with the following therapeutic schedules: (1) streptomycin, 1 gm. daily for 42 days; (2) streptomycin, 1 gm. daily, paraaminosalicylic acid, 8 gm. daily, for 120 days; (3) streptomycin, 1 gm. daily, paraaminosalicylic acid, 15 gm. daily, for 120 days; (4) streptomycin, 1 gm. daily, paraaminosalicylic acid, 12 gm. daily, for 90 days, and (5) streptomycin, 1 gm. daily, paraaminosalicylic acid, 12 gm. daily, for 120 days. Streptomycin sensitivity tests were recorded at 60, 90, and 120 days after the start of treatment. There was a decided reduction in the number of patients showing tubercle bacilli that were resistant to streptomycin when combined therapy with paraaminosalicylic acid was employed.

Gastroenterology, Baltimore

17:479-628 (April) 1951

Infarcts of Liver. K. R. Woolling, A. H. Baggenstoss and J. F. Weir. —p. 479.
Post-Bulbar Duodenal Ulceration. W. M. Lonergan and A. Kahn Jr. —p. 494.
Abdominal Aortic Aneurysm with Rupture into Duodenum: Report of Eight Cases. A. E. Hirst Jr. and J. Affeldt.—p. 504.
Pharyngeal Anesthesia for Gastroscopy and Esophagoscopy—New Simplified Technique. S. Selesnick.—p. 515.
Amebiasis in Veterans: IV. Results of Treatment with Aureomycin and Chloramphenicol. H. C. Conn.—p. 517.
*Jaundice in Chronic Hepatitis (Cirrhosis of Liver). W. E. Ricketts. —p. 523.
*Ineffectiveness of Enterogastrone on Severe Chronic Peptic Ulcer in Man. A. Wollum and H. M. Pollard.—p. 535.
Use of Testosterone Propionate Following Extensive Resection of Small Intestine. I. J. Pincus.—p. 541.
Effect of Terramycin on Intestinal Flora. J. A. Rivera and V. M. Sborov. —p. 546.
Oral Banthine, Effective Depressor of Gastrointestinal Motility. M. J. Lepore, R. Golden and C. A. Flood.—p. 551.
Some Effects of Banthine on Pancreatic Secretion. D. Annis and G. A. Hallenbeck.—p. 560.
Studies on Bromsulfalein Excretion: I. Results of Successive Daily Bromsulfalein Tests and Effects of Galactose on Bromsulfalein Excretion. E. S. Cohen, T. L. Althausen, K. Uyeyama and H. Treager.—p. 572.

Jaundice in Chronic Hepatitis.—In order to determine the significance and pathogenesis of jaundice in portal cirrhosis, Ricketts studied 28 consecutive patients, three children and 25 adults (22 males and six females) with pronounced jaundice by simultaneous clinical and histological observation and by conventional biochemical determinations. A previous history of alcoholism was detected in 13 of the 28 patients and an insidious onset of jaundice in 21. The following findings, occurring singly or in combination, were significant in the diagnosis of chronic hepatitis: collateral circulation of the abdomen, spider nevi, telangiectasis, hard or nodular liver, and varicose veins of the

esophagus. Punch biopsy of the liver was the most important single diagnostic procedure used, since it served to demonstrate not only liver fibrosis but also changes of the liver parenchyma. Jaundice in patients with cirrhosis represents active parenchymatous liver disease and as such cannot be regarded as a manifestation of "compensated cirrhosis." Pathologically there were active changes of the liver parenchyma that varied considerably in type and severity. There was no relation between jaundice and the degree or type of fibrosis. Jaundice appeared as a slight, transitory, almost unnoticed episode in the course of chronic hepatitis, or most commonly it was accompanied by other symptoms and findings of liver failure, such as edema, ascites, and encephalopathy. Jaundice is to be considered a very serious sign; nine of the 28 patients died during the episode of jaundice, eight of liver failure and one of liver failure and gastrointestinal bleeding. The presence of jaundice contraindicates abdominal surgery. The importance of medical management of portal cirrhosis with jaundice is emphasized, since even patients with severe liver failure can recover clinically. Recovery is accompanied by disappearance of degenerative features, regeneration of the liver parenchyma, and pronounced improvement of parenchymal function.

Ineffectiveness of Enterogastrone in Peptic Ulcer.—Thirty-four patients, 32 men and two women with chronic peptic ulcer, were treated with enterogastrone for prolonged periods. Enterogastrone was given orally to 19 patients, of whom two were given doses of 6 gm. daily for an average of 6.5 months, five were given 8 gm. daily for an average of 11.5 months, and 12 were given 16 gm. daily for an average of 7.2 months. Intramuscular injections of 200 mg. of enterogastrone were given to 15 patients six times a week for an average of 5.5 months. Only eight of the 34 patients, with an average duration of the ulcer history of 14.2 yr., were significantly improved, as shown by decreased recurrence rate, absence of complications, or lack of need for surgery. Of these eight patients, three had received parenteral enterogastrone, four had received 16 gm. of oral enterogastrone daily, and one had received 8 gm. of oral enterogastrone daily. Twenty-six patients had an increase in recurrences, more intense symptoms, or ulcer complications such as obstruction, hemorrhage, or perforation. Eighteen patients underwent surgical treatment because of ulcer complications or intractable symptoms. Prolonged oral administration of enterogastrone in doses of 16 gm. daily or of intramuscularly administered enterogastrone did not significantly alter the rate of gastric secretion, free hydrochloric acid content of gastric juice, or pepsin content of gastric juice in the authors' patients. Gastric motility was also not altered.

Indiana State Medical Assn. Journal, Indianapolis

44:353-432 (May) 1951

*Cardiotoxic Action of Intravenous Procaine. F. S. Carter and J. L. Eisaman.—p. 369.
Surgical Treatment of Hyperthyroidism. L. H. Kornafel.—p. 374.
Are Shoe Fitting Fluoroscopes a Health Hazard to the Public in Indiana? H. E. Bumstead and L. W. Spolyar.—p. 376.
Shoulder Pinning for Neck Fractures of Humerus. F. E. Hagie.—p. 379.
Clinical Management of Peptic Ulcers in General Practice. O. R. Bowen.—p. 381.

Cardiotoxic Action of Intravenously Administered Procaine.—Electrocardiographic studies were made on 42 patients in connection with procaine infusions. A total of 257 electrocardiograms were made in connection with 263 infusions. The tracing made prior to the first infusion served as the control. In most cases procaine infusions were given daily; others received infusions on alternate days. Treatment was given until either an excellent therapeutic result was obtained or further infusions failed to improve the condition. Analysis of the electrocardiograms revealed P-wave alterations in four cases, prolongation of the P-R interval in 5 cases, a decrease in 17 cases and an increase in two cases in the QRS voltage, and a T voltage decrease in 16 cases and an increase in eight cases. Twelve patients showed no change following repeated infusions; of the others, some showed one and others several changes. In two cases the changes were sufficiently severe to justify cessation of treatment. The duration of the changes was variable and could not be correlated with their extent. In general, the changes were gone in 24 hr., but in several patients they persisted longer than three days. The following conclusions are drawn on the basis of the described observations: Intravenously administered procaine has a depressant action on both the myocardium and the conduction system. This action is beneficial in ventricular arrhythmias but does not occur in auricular arrhythmias. Intravenously administered procaine should be used with caution in persons with heart disease; electrocardiograms should be made before and during treatment.

Journal of Applied Physiology, Washington, D. C.

3:637-690 (May) 1951. **Partial Index**

Effect of High Carbon Dioxide and of Low Oxygen Concentration on Fusion Frequency of Flicker. E. Simonson and P. Winchell.—p. 637.
Oximetric Determinations of Cardiac Output Responses to Light Exercise. W. Sleator Jr., J. O. Elam, W. N. Elam and H. L. White.—p. 649.
Diurnal Variations in Body Temperatures. H. C. Mellette, B. K. Hutt, S. I. Askovitz and S. M. Horvath.—p. 665.
Relation of Specific Gravity to Body Build in Group of Healthy Men. C. W. Dupertuis, G. C. Pitts, E. F. Osserman and others.—p. 676.

Journal of Aviation Medicine, St. Paul

22:87-176 (April) 1951

High Altitude-High Velocity Flying with Special Reference to Human Factors: III. Bare Skin Hazard from Frostbite in Escape from Aircraft. A. P. Webster and H. A. Smedal.—p. 89.
Human Time Factor in Flight: II. Chains of Latencies in Vision. H. Strughold.—p. 100.
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Comparative Experimental Studies of Effects of Simulated High Altitudes on Five Vertebrates. B. Metz.—p. 132.
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Pilot Candidate Selection Research Project. V. W. Lyon.—p. 152.
Pick-Up and Amplifier for the Ballistocardiograph. C. V. Pestel and J. P. Marbarger.—p. 156.

Journal of Bacteriology, Baltimore

61:379-526 (April) 1951. **Partial Index**

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Adaptation of Quality Control Chart Methods to Bacterial Vaccine Potency Testing. H. C. Batson, M. Brown and M. Oberstein.—p. 407.
Effect of Aeration on Growth of Aerobacter Aerogenes and Escherichia Coli, with Reference to Pasteur Mechanism. S. Dagley, E. A. Dawes and G. A. Morrison.—p. 433.
Improved CO_2 Cabinet for Low Temperature Storage of Infectious Agents with Gaseous CO_2 Excluded from Specimen Compartment. F. L. Horsfall Jr. and H. S. Ginsberg.—p. 443.
Electron Microscopic Observations of Flagellation in Some Species of Genus Treponema Schaudinn. J. H. L. Watson, J. J. Angulo, F. León-Blanco and others.—p. 455.
Utilization of Nitrogen in Various Compounds by Clostridium Pasteurianum. E. D. Rosenblum and P. W. Wilson.—p. 475.
Nitrogen Fixation and Hydrogenase in Various Bacterial Species. E. S. Lindstrom, S. M. Lewis and M. J. Pinsky.—p. 481.
Adaptations in Growth Factor Requirements for Lactic Acid Bacteria. V. H. Cheldelin and A. P. Hygaard.—p. 489.
Growth of Bacteria in Charcoal-Cellophane System. A. N. Gorelick, D. D. Mead and E. H. Kelly.—p. 507.

61:527-648 (May) 1951. **Partial Index**

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Prophylaxis of Experimental Gas Gangrene in Mice. W. I. Taylor and M. V. Novak.—p. 571.
Influence of Vitamin B_{12} on Oxidation by Mutant Strain of Escherichia Coli. E. L. Oginsky, P. H. Smith, N. E. Tonhazy and others.—p. 581.
Action of Streptomycin: V. Formation of Citrate. W. W. Umbreit, P. H. Smith and E. L. Oginsky.—p. 595.
Action of Streptomycin on Respiration of Streptomyces Griseus. E. L. Oginsky, P. H. Smith and W. W. Umbreit.—p. 639.
New Bacteriophage Active Against Lactatic Streptococcus. K. I. Williamson and W. S. Bertaud.—p. 643.

Journal Clin. Endocrinology, Springfield, Ill.

11:455-558 (May) 1951

Metabolic Effects of ACTH upon Pre-Existing Diabetes Mellitus. S. S. Fajans, L. H. Louis and J. W. Conn.—p. 455.
Urinary Prolactin Excretion in Man. R. L. Coppedge and A. Segaloff. —p. 465.
Sexual Trends in Female Pseudohermaphrodism. F. Hinman Jr.—p. 477.
Treatment of Adolescent Acne with Topical Application of Estrogens. M. J. Whitelaw.—p. 487.
Scintillation Counter in Clinical Studies of Human Thyroid Physiology Using I^{131}. H. C. Allen Jr., R. L. Libby and B. Cassen.—p. 492.
*Irradiation of Pituitary in Treatment of Malignant Exophthalmos. W. H. Beierwaltes.—p. 512.
Growth Hormone and Experimental Diabetes. F. G. Young.—p. 531.
Pituitary Necrosis and Diabetes Mellitus. E. R. Marzullo and M. B. Handelsman.—p. 537.

Irradiation of Pituitary in Malignant Exophthalmos. — The initial symptoms of "malignant" exophthalmos are usually orbital, retro-orbital, or frontal pain, burning, lacrimation, photophobia, and, less commonly, diplopia. The eyes become proptosed; the lids swell, and edema and fluid appears under the conjunctivas. Conjunctival sacs may protrude between the lids or hang down over the cheeks. When the patient loses his ability to close the eyes while asleep, exposure keratitis, corneal ulceration, panophthalmitis, surgical loss of the orbit, or death may follow. Decrease in protrusion of the eyes is rarely brought about by lowering of the basal metabolic rate when it is elevated or by the administration of desiccated thyroid when it is not. Surgical decompression of the orbit is effective in this condition, but this is a relatively formidable procedure. Roentgen therapy directed to the pituitary was used in 10 patients with malignant exophthalmos and in one patient with progressive exophthalmos. These patients were selected for treatment because their eye status became worse during an average control period of 10.7 months, during which time they were treated with desiccated thyroid. Seven of the 10 patients with malignant exophthalmos and the one patient with progressive exophthalmos had significant recession in exophthalmos. Only one of the three nonresponsive patients showed a significant increase in exophthalmos. Transient epilation resulted in all patients, and there were occasional headaches during the first few weeks after treatment. Patients who responded showed recession at an average interval of three months and maximum recession at an average interval of 12 months after roentgen irradiation.

Journal of Experimental Medicine, New York

93:415-512 (May) 1951

Role of Alkaline Phosphatase in Osteogenesis. R. S. Siffert.—p. 415.
Age of Host and Other Factors Affecting Production with Urethane of Pulmonary Adenomas in Mice. S. Rogers.—p. 427.
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Joint Action of Chemical Carcinogen and Neoplastic Virus to Induce Cancer in Rabbits: Results of Exposing Epidermal Cells to Carcinogenic Hydrocarbon at Time of Infection with Shope Papilloma Virus. S. Rogers and P. Rous.—p. 459.
Studies on Mechanism of Shwartzman Phenomenon: Certain Factors Involved in Production of Local Hemorrhagic Necrosis. C. A. Stetson Jr.—p. 489.
Lymphoid Lesions in Poliomyelitis. S. C. Sommers, J. C. Wilson and F. W. Hartman.—p. 505.

Journal of Infectious Diseases, Chicago

88:105-206 (March-April) 1951. **Partial Index**

Effects of Protein Deficiency on Resistance of Rats to Infection with Spirochetes of Relapsing Fever. K. Guggenheim, E. Buechler-Czaczkes and S. Halevy.—p. 105.
Human Carrier of Psittacosis Virus. K. F. Meyer and B. Eddie.—p. 109.
Dynamic Aspects of Nitrogen Metabolism of Plasmodium Gallinaceum in Vivo and in Vitro. N. B. Groman.—p. 126.
Use of Gastric Mucin to Lower Resistance of Laboratory Animals to Systemic Fungus Infections. R. E. Strauss and A. M. Kligman.—p. 151.
Studies on Plasmodium Gallinaceum in Vitro. D. J. Taylor, J. Greenberg, E. S. Josephson and A. P. Ray.—p. 158.
Toxic and Allergic Manifestations Caused by Tissues and Fluids of Ascaris. J. F. A. Sprent.—p. 168.
Response of Syrian Hamster to Oral Administration of Modified Newcastle Disease Virus. R. L. Reagan, D. M. Schenck, H. O. Werner and A. L. Brueckner.—p. 178.
Search for Poliomyelitis Virus in House Dust and Study of Effects of Drying on Viability of Virus. H. K. Faber, L. Dong and R. J. Silverberg.—p. 180.

Journal of International College of Surgeons, Chicago

15:531-668 (May) 1951

Diagnosis of Surgical Diseases of Spleen. T. H. Bate.—p. 531.
Study of Breast Secretion for Detection of Intramammary Pathologic Change and of Silent Papilloma. D. Jackson, D. A. Todd and P. L. Gorsuch.—p. 552.
Endocrine Therapy in Gynecologic Practice. J. Kotz and M. S. Kaufman. —p. 569.
Polyethylene Intubation in Reimplantation of Oviducts. J. I. Perl.—p. 576.
Personal Evaluation of Vagotomy in Treatment of Peptic Ulcer. K. C. Sawyer.—p. 585.
Pilonidal Sinus: Etiology of Recurrence. H. R. Grau.—p. 591.
Recurrent Inguinal Hernia. D. P. Hall.—p. 605.
Management of Eye Injuries by the General Surgeon in Absence of an Ophthalmologist. F. L. P. Koch.—p. 614.
Foreign Bodies in Hepatic Ducts: Review of Literature. M. Thorek. —p. 624.
Eosinophilic Variations After Irradiation of Adrenal Glands. M. A. Manzanilla and M. A. Manzanilla Jr.—p. 628.
Berylliosis and Silicosis. R. Pomeranz and H. A. Brodkin.—p. 633.
*Retropubic Prostatectomy: Modification of Millin Operative Technic (1945). C. M. Carruthers.—p. 639.
Problem of Thyroid Gland from Standpoint of Anesthesiologist. J. S. Lundy.—p. 646.
Concept of Mobilization and Its Relation to Poliomyelitis. H. E. Billig Jr.—p. 649.

Retropubic Prostatectomy: Modification of Millin Technique.— This is a report of Carruthers' modification of Millin's technique of prostatectomy and is based on a series of 53 consecutive prostatectomies, performed during the last two and one-half years. This modification allows better exposure, anatomic dissection, and control of bleeding, with minimal shock to the patient. It is applicable in all types of prostatic obstruction and makes this field available to the general surgeon or urologist who is not trained in the highly specialized field of transurethral resection. The gland is completely removed, and there is no tendency to stricture formation. Postoperative urethrograms show the prostatic cavity to be confluent with the urinary bladder. Nursing and hospital care are minimal and well suited to a general hospital. Confinement to bed has been shortened, with an early return to normal micturition. Sexual function has not been disturbed.

Journal of Investigative Dermatology, Baltimore

16:295-378 (May) 1951

Injection and Ingestion Test in Cross-Sensitization to Para Group. E. Sidi and S. Dobkewitch-Morrill.—p. 299.
Spectrophotometry of Living Human Skin in Ultraviolet Range. E. A. Edwards, N. A. Finklestein and S. Q. Duntley.—p. 311.
Effects of ACTH and Cortisone on Certain Diseases and Physiologic Functions of Skin: I. Effects of ACTH. M. B. Sulzberger, G. C. Sauer, F. Herrmann and others.—p. 323.
Cyst Formation, Acneform Lesions and Hair Growth Following Intradermal Injection of Staphylococci in Sensitized Rabbits. J. G. Hopkins, J. T. Weld and W. M. Huber.—p. 339.
Pituitary-Adrenocortical Functioning in Patients with Atopic Dermatitis. T. H. Sternberg, P. Butler and M. C. Zimmerman.—p. 355.
Pain Threshold Measurements on Human Skin Following Application of Topical Analgesics. J. D. Hardy, C. B. Potelunas and M. D. Meixner. —p. 369.

Journal-Lancet, Minneapolis

71:169-214 (May) 1951

Congenital Atresia of Esophagus and Intestine: With Report of Cases. G. A. Dodds, L. G. Pray, V. G. Borland and W. E. Le Bien.—p. 169.
Congenital Duodenal Obstruction: With Report of Two Cases. R. E. Dyson.—p. 174.
Surgical Relief of Atelectasis in the Newborn. E. B. Berglund, W. P. Eder, O. Wyatt and T. Chisholm.—p. 179.
Hydronephrosis in Children. F. D. Naegeli.—p. 182.
Herpes Zoster with Motor Involvement. R. B. Tudor.—p. 184.
Social Isolation and Feral Behavior in a Child of Three. W. Fleeson. —p. 185.
Central Nervous System Sequelae Following Injections of Pertussis Vaccine. R. B. Tudor.—p. 188.
Progressive Diaphyseal Dysplasia: With Report of Case. W. E. Le Bien and C. Heilman.—p. 189.
Polyostotic Fibrous Dysplasia: With Report of Case. E. S. Krug and H. R. Glenn.—p. 193.
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Journal of Pediatrics, St. Louis

38:537-666 (May) 1951

Tonsillectomy and Poliomyelitis: I. Studies on Incidence of 1949. M. Siegel, M. Greenberg and M. C. Magee.—p. 537.
Id.: II. Frequency of Bulbar Paralysis, 1944-1949. M. Siegel, M. Greenberg and M. C. Magee.—p. 548.
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Treatment of Infantile Diarrhea with New Combination of Antibiotics. E. R. Kadison and M. P. Borovsky.—p. 576.
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Treatment Program for Chronic Sinobronchitis. W. C. Price and V. B. Binns.—p. 597.
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Function of Recovery Nursery in Large Maternity Hospital. A. Bloxsom. —p. 618.

J. Pharmacology & Exper. Therap., Baltimore

101:317-404 (April) 1951

Effects of Potassium, Ammonium, Calcium, Strontium and Magnesium on Electrogram and Myogram of Mammalian Heart Muscle. S. Garb. —p. 317.
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Effect of Certain Compounds on in Vitro Synthesis of Urea and Its Precursors. G. R. McKinney.—p. 345.
Some Pharmacologic Properties of m-Tolyloxyacetamidine Hydrochloride, a Cardiotonic Drug. B. N. Craver, A. Cameron and J. Smith.—p. 353.
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Effect of 1-Hydrazinophthalazine (C-5968) and Related Compounds on Cardiovascular System of Dogs. H. A. Walker, S. Wilson, E. C. Atkins and others.—p. 368.
Adrenergic Blocking Action of Phenoxyethyl Analogues of Dibenamine. M. Nickerson and G. M. Nomaguchi.—p. 379.

Journal of Thoracic Surgery, St. Louis

21:433-542 (May) 1951

Lung Function Studies: VII. Effect of Pneumonectomy on Respiratory Dead Space. W. S. Fowler and W. S. Blakemore.—p. 433.
Obstructive Emphysema with Defect of Anterior Mediastinum: Report of Case. J. E. Lewis and W. J. Potts.—p. 438.
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Pathology of Experimentally Produced Lye Burns and Strictures of Esophagus. L. H. Bosher Jr., T. H. Burford and L. Ackerman.—p. 483.
Cervical Esophagostomy. C. T. Klopp.—p. 490.
Mobilization of Arch of Aorta in Order to Reach Bifurcation of Trachea. O. Monod, G. Babou, E. Kateb and S. Ghazi.—p. 492.
Tracheotomy, Useful Procedure in Thoracic Surgery, with Particular Reference to Its Employment in Crushing Injuries of Thorax. B. N. Carter and J. Giuseffi.—p. 495.
Coarctation of Aorta at Level of Diaphragm Treated Successfully with Preserved Human Blood Vessel Graft. E. J. Beattie Jr., F. N. Cooke, J. S. Paul and J. A. Orbison.—p. 506.
Effective Method for Bronchial Closure. R. A. Rasmussen.—p. 513.
Bronchial Yardstick. E. B. Emerson Jr.—p. 516.
Clinically Indeterminate Pulmonary Fibrosis: Pathologic Study. H. D. Peabody Jr., H. J. Moersch and J. E. Edwards.—p. 519.
*Oxygen Administration: Emergency Method When Intratracheal Tube Fails with Chest Open. O. C. Brantigan.—p. 532.
Bronchial Adenoma: Analysis of 26 Cases. N. C. Delarue.—p. 535.

Oxygen Administration When Intratracheal Tube Fails.—Brantigan describes a new technique of emergency oxygen administration for use when an intratracheal tube slips out during anesthesia. A no. 17 intravenous needle, which is attached by rubber tubing to an oxygen tank, is inserted into the bronchus. The assistant holds the needle in place, and the operator controls the flow of oxygen. The no. 17 needle acts as a safety mechanism, since it tends to prevent an excessive flow. The escape of oxygen from the glottis aids in reinsertion of the intratracheal tube, which can be done without haste. It is suggested that the oxygen tank and sterile rubber tubing with needle attached be available in the operating room.

Journal of Urology, Baltimore

65:497-692 (April) 1951. **Partial Index**

Experimental Studies on Male Reproductive System. C. R. Moore. —p. 497.
Lower Nephron Syndrome in Children. H. I. Riddell.—p. 513.
Progressive Synergistic Gangrene of Skin Following Incision and Drainage of Renal Cortical Abscess. W. F. Welton.—p. 519.
Ureteral Calculi: Experiences in Looped Catheter Management. M. Ellik and L. A. Newton.—p. 532.
Glandular Tumors of Bladder. W. M. Coppridge, L. C. Roberts and D. A. Culp.—p. 540.
Preparation, Operative Technique and Postoperative Management of Uretero-Intestinal Anastomosis. C. Rusche.—p. 550.
*Cystectomy and Urinary Diversion in Carcinoma of Bladder. G. J. Thomas, D. C. Malcolm and A. J. Bischoff.—p. 553.
Hydrodynamics of Upper Urinary Tract After Mucosal Ureterosigmoidostomy: Case Report. J. M. Whisenand and V. Moore.—p. 564.
Complications and End Results Following Uretero-Intestinal Anastomosis. J. J. Crane, F. A. Bennetts, G. H. Gummess and J. F. Crane.—p. 571.
*Substitute Bladder and Urethra, Using Cecum as Bladder and Ileum as Urethra. J. W. Merricks, R. K. Gilchrist, H. Hamlin and I. T. Rieger. —p. 581.
Congenital Diaphragm and Fistula of Penile Urethra. H. C. Tseng. —p. 590.
Conservative Treatment of Highly Obstructive Cancer of Prostate. R. B. Mullenix and R. J. Prentiss.—p. 608.
Malignant Disease of Testicle. J. C. Kimbrough and J. C. Denslow. —p. 611.
Sources of Danger in Urinary Extravasation: Hazard of Perforation During Transurethral Prostatectomy. H. M. Weyrauch, R. A. Peterfy and J. L. Goebel.—p. 615.
March Hemoglobinuria. R. T. Plumb.—p. 655.
Urogenital Aspects of Coccidioidomycosis: Review of Literature and Report of Two Cases. J. G. Rohn, J. C. Davila and T. E. Gibson. —p. 660.
*Effect of Combined Therapy on Emergence of Drug Resistant Bacteria in Urinary Tract Infections: Observations on Origin of Resistant Strains. L. J. Kimmelman, H. H. Zinsser and M. Klein.—p. 668.

Cystectomy and Urinary Diversion in Carcinoma of Bladder.—Cystectomy was done for cancer of the bladder in 36 patients at two Veterans Administration hospitals, one of which was a tumor center. A total of 325 patients with carcinoma of the bladder were observed at these hospitals. In patients who underwent cystectomy an attempt at removal of cancer of the bladder had been delayed an average of three and a half years. From their study of these cases, the authors made the following conclusions: Gross hematuria may not be the first symptom of cancer of the bladder, but, since most tumors of the bladder produce blood and other cells in the urinary sediment long before gross hematuria occurs, such findings should suggest a complete urologic examination. The cystoscopist, who can view the entire bladder, can make a more accurate diagnosis of malignant change than the pathologist who examines only a bit of tissue removed transurethrally. Because of frequent errors in the study of biopsy specimens, surgical exploration of the bladder and the surrounding structures should be done before deciding on a cystectomy. At this exploratory operation the diversion of the urinary stream into the bowel or into the skin can be undertaken. Cystectomy should be used only when the degree of malignancy is no greater than grade 2 and when the cancer is confined to the lumen of the bladder. Metastases occur early and may be widespread with any degree of malignancy. Diversion of the urinary stream into the sigmoid or through the skin is the best palliative treatment. Implantation into the skin is the safest method. In this series there was a high mortality rate following cystectomy. Two patients lived four years and 11 months, and one lived five years and six months. Eight (29%) lived more than a year. One lived more than two years. The average survival was 10 months. There were local recurrences in many cases, and metastases to the bones also occurred frequently.

Substitute Bladder and Urethra.—In the treatment of certain tumors of the rectum, uterus, bladder, and prostate, in certain congenital anomalies and in conditions such as extensive vesicovaginal and rectovaginal fistulas, it may be highly desirable to divert the urinary stream above the bladder. Numerous operations have been designed, but each has limitations. In the technique described in this paper the new bladder is made from the isolated terminal ileum, cecum, and ascending colon. Bowel continuity is reestablished by ileocolostomy, and the ureters are transplanted to the new bladder. The terminal ileum is brought out to the abdomen to serve as the urethra and an indwelling catheter is left in the new bladder. Finally, the patient is taught how to catheterize himself often enough to prevent overflow

leakage. This arrangement has the following advantages: (1) There is no contamination by the fecal stream. (2) The ureteral anastomoses are easily made and can be covered by peritoneum. (3) A cystoscope can be passed to the new bladder and the ureteral orifices can be dilated or otherwise treated if need be. (4) No device is needed to prevent leakage. (5) There is no limitation of radical surgery when dealing with malignant lesions. In animal experiments and in the follow-up study of human patients the authors encountered no real contraindication to this procedure.

Combined Therapy and Drug Resistance.—This study shows that the therapeutic effect of combined antibacterial agents is correlated with the inhibition of drug resistance. A total of 37 cases, including both uncomplicated urinary tract infections (e. g., cystitis, pyelonephritis, and urethritis) and those complicated by pathological changes, such as calculi, strictures, and obstructions, were studied. The patients received the following daily dosage for a total of seven days: streptomycin, 1.0 gm., intramuscularly; penicillin, 200,000 units, intramuscularly; sulfadiazine, 2.0 gm., orally. Sterile urine cultures were obtained in 16 cases, 14 of which were uncomplicated urinary tract infections. Therapy was bacteriologically unsuccessful in 21 cases, 19 of which were characterized by some complication. In these 19 cases, bacteria resistant to all three drugs regularly emerged. A study of resistant strains in the urine and feces of seven patients strongly indicated that many of the resistant strains in the urine had their origins in the fecal flora of the patient. Good clinical response was obtained in virtually all the cases in spite of persistent bacilluria. The combined drugs produced no evidence of toxicity. If combined therapy is to be of permanent value, efforts must be made to remove the cause of the obstruction or complication.

Kansas Medical Society Journal, Topeka

52:209-256 (May) 1951

Infantile Cortical Hyperostoses. J. F. Bowser.—p. 209.
Intracranial Aneurysms. J. Hibbard, R. L. Drake and L. E. Vin Zant.—p. 213.
Abdominal Distention: Prevention and Treatment with Stigminene Bromide. L. J. Hanis and C. D. Kobler.—p. 216.
Diagnosis and Treatment of Subdural Hematomas in Infants. J. R. Patterson.—p. 218.

Medicine, Baltimore

30:81-196 (May) 1951

Benign Intramural Tumors of Stomach: Review with Special Reference to Gross Pathology. E. D. Palmer.—p. 81.
Ion Exchange Resins in Edema. E. W. McChesney, W. Dock and M. L. Tainter.—p. 183.

Michigan State Medical Society Journal, Lansing

50:441-552 (May) 1951

Obstruction of Common Bile Duct by Stones. W. H. Cole.—p. 481.
Allergic Myalgia. T. G. Randolph.—p. 487.
Common Geriatric Dermatoses. E. W. Netherton.—p. 495.
Kala-Azar: Report of Case and Review of Recent Literature. T. A. McInerney and R. E. Ryde.—p. 501.
Review of Endometriosis: Summary of 32 Cases of Hurley Hospital. J. W. Thompson.—p. 504.
Congenital Paralysis of Inferior Oblique. H. W. Brown.—p. 509.

Military Surgeon, Washington, D. C.

108:369-466 (May) 1951. **Partial Index**

Civil Defense Planning. N. C. Kiefer and R. H. Flinn.—p. 372.
Definitive Treatment of Pulmonary Tuberculosis: General Therapeutic Principles as They Apply to Various Types of Chest Lesions. C. W. Tempel and J. H. Forsee.—p. 375.
Postgraduate Medical Education Program of the United States Air Force Medical Service. R. B. Jones.—p. 408..
*Experimental Use of Bone Marrow in Acute Radiation Injury. J. M. Talbot and E. A. Pinson.—p. 412.
Division Combat Medical Service. T. Fox.—p. 427.
Potentialities of Biological Warfare Against Man—Epidemiological Appraisal. A. D. Langmuir.—p. 429.

Bone Marrow in Radiation Injury.—This report deals with the use of bone marrow in animals severely injured by acute, whole body exposure to roentgen irradiation. The experimental approach was based on the fact that in this type of injury there is extensive bone marrow damage with extreme depression of hematopoiesis and on the hypothesis that this results in an unusual demand for the products of marrow metabolism. Preliminary studies of bone marrow homotransplants in rats with marrow depressed by benzene injections and by whole body roentgen irradiation suggested that the transplants would survive in a reasonably healthy condition for an approximate minimum of four weeks. In the main experiment one group of white rats exposed to 500 r total body irradiation was given one intramuscular injection of marrow and a similar group two injections of marrow three to four days apart. Another series of rats received intrahepatic marrow homotransplants following single, whole body exposures to 700 r. A control group not operated on and a special control group the hind limbs of which were two-thirds to three-fourths shielded by 1.5 cm. of lead were simultaneously studied. There was no apparent benefit in terms of survival and postirradiation total leukocyte counts in the case of the marrow-transplanted group; however, the survival of these animals, each of which experienced general anesthesia, laparotomy, and injection into the liver, was at least as good as that of the controls not operated on. The shielded group had a mean survival time 2.4 times that of both the animals with transplants and the control animals.

Minnesota Medicine, St. Paul

34:389-516 (May) 1951

Fluid and Electrolyte Balance in Intestinal Obstruction. F. A. Coller and R. E. L. Berry.—p. 414.
*Primary Intestinal Anastomosis. I. S. Ravdin and W. DeMuth Jr.—p. 419.
Prognostic Factors in Colonic Malignancy. C. F. Dixon.—p. 424.
Pulmonary and Extrapulmonary Tuberculosis with Discussion of Host Response to Tuberculosis. D. T. Smith.—p. 428.
Problems of Rural Medical Care. F. D. Murphy.—p. 433.
Rheumatic Pneumonia. R. B. Tudor and R. R. Kling.—p. 437.
Operating Room Cholangiograms. A. Zierold.—p. 439.
Practical Points in Allergy. J. H. Shaffer.—p. 442.

Primary Intestinal Anastomoses.—This and other papers in this issue pay tribute to Owen H. Wangensteen for his work in relieving gastric and intestinal distention by intubation. Intestinal intubation as introduced by Wangensteen and extended by others has reduced the morbidity and mortality rates of many diseases and procedures. Ravdin and DeMuth say that with its help bowel resection and primary anastomosis has become a safe operation in many patients. During the past five years there has been a steady increase in the number of primary anastomoses following bowel resection, regardless of location of the lesion. The control of distention is the clue to successful preparation of the bowel for primary anastomosis. The authors report results obtained in the five-year period between 1946 and 1950 inclusive in 394 patients in whom primary intestinal anastomoses accompanied by intubation and suction drainage were the sole source of decompression. Intestinal resections were done in all but 38 of the reported cases. The over-all mortality rate was 3.8%. Primary anastomosis following resection of bowel segments or used alone in bypassing inoperable lesions, is a safe, rational procedure that saves the patient both time and prolonged incapacity. In the treatment of cancer it in no way modifies the radical approach to the eradication of the disease.

Neurology, Minneapolis

1:179-260 (May-June) 1951. **Partial Index**

Importance of Neuroanatomy in Field of Neuropathology. C. Vogt and O. Vogt.—p. 205.
*Succinate Therapy in Multiple Sclerosis. R. B. Aird.—p. 219.
Diabetic Neuropathy and Its Prognosis. S. H. Epstein.—p. 228.
Aphasia in Deaf Mute. L. L. Tureen, E. A. Smolik and J. H. Tritt.—p. 237.
Medical Treatment of Psychomotor Epilepsy. J. K. Merlis.—p. 245.

Succinate Therapy in Multiple Sclerosis.—Sodium succinate was administered intravenously (20 cc. of a 30% solution) to 10 patients with multiple sclerosis and orally (4 to 5 gm. of powder in 1 oz. [30 cc.] of water three times a day) over prolonged periods to 22 patients with multiple sclerosis. Subjective improvement was reported in 80%, and objective improvement was observed in approximately two-thirds of the patients by both methods of treatment. Follow-up observations on patients treated orally with succinate suggested that approximately one-half have benefited. Beneficial effects consisted of (1) improvement in dyssynergia, ataxia, dysmetria, balance, and gait, (2) improvement

in the flexor withdrawal reflex and clonus in some patients, and (3) subjective improvement in the appreciation of vibration and objective improvement in position sense and skin writing. Toxic effects were observed in one patient treated intravenously and in six patients given oral succinate. The toxic reactions to oral administration consisted of gastrointestinal and genitourinary effects and were not alarming. Three of these patients were able to continue treatment with benefit on reduced doses. Deterioration on discontinuance of the succinate was observed in four instances, and all these patients improved when therapy was resumed. Recurrent episodes have been observed in three patients. The rationale for the use of sodium succinate in multiple sclerosis is discussed.

New England Journal of Medicine, Boston

244:699-742 (May 10) 1951

American Physician and His Opportunities for Increased Service Through Public Health. J. S. Simmons.—p. 699.
*Acid Débridement of Burns with Phosphoric-Acid Gel. R. J. Schweitzer and J. T. Bradsher Jr.—p. 705.
Delirium and Coma Precipitated by Oxygen in Bronchial Asthma Complicated by Respiratory Acidosis. H. D. Beale, I. W. Schiller, M. H. Halperin and others.—p. 710.
Survey of Present-Day Treatment of Diabetes Mellitus. S. B. Beaser. —p. 714.
Medicine as Social Instrument: Medical Social Service. I. M. Cannon. —p. 717.

Acid Débridement of Burns.—Attempts have been made to find some agent that would selectively digest or remove the necrotic slough that follows severe burns. Connor and Harvey in 1944 used pyruvic acid starch paste as such an agent, but since this substance had certain disadvantages, other methods were tested. Only phosphoric acid was equal to pyruvic acid in efficiency of slough removal and low degree of irritation. At the Boston City Hospital phosphoric acid (85%) was used as the débriding agent. This was added to a water-soluble vehicle to form a stable 0.7 molar (5%) phosphoric acid gel (pH, 1.02). Immediately after application of the phosphoric acid gel a burning sensation was felt, which lasted from 15 to 30 min. Premedication with small amounts of meperidine (demerol®) hydrochloride or morphine relieved this discomfort. At the first dressing, parallel incisions were made in the slough, to increase the accessibility of the agent to the site of its activity. A dam of petrolatum gauze was placed about the burn to prevent the mixture from seeping away; later, felt strips impregnated with petrolatum were found most satisfactory to accomplish this. The wound was covered with phosphoric acid gel, and petrolatum strips and fluffed gauze were applied over this. Dressings were changed daily or every other day. When the cleavage plane became apparent the separation of partially débrided and undermined areas could be hastened mechanically. Epithelial islands were apparently not damaged by the débriding agent. Areas with first, second, and third degree burns could be treated beneath the same dressing with safety. After débridement, the wounds were dressed with a weak chlorinate solution until the operating schedule permitted skin grafting or, in the case of superficial second degree burns, until healing had occurred. Phosphoric acid gel was employed in 11 cases and proved a simple, safe, selective, and rapid method of débridement. The course of six of these patients is described.

New Jersey Medical Society Journal, Trenton

48:137-182 (April) 1951

Trends in Nursing Education and Practice. R. A. Schaaf.—p. 139.
Diabetic Ketosis. P. B. Ferrary.—p. 141.
Very Low Sodium Diet: Both Palatable and Variable. G. G. Ornstein. —p. 145.
The New Jersey Formulary—Sixth Edition. T. D. Rowe and J. L. Varriano.—p. 150.
Hematometria from Complete Amputation of Cervix During Labor. N. Karshmer.—p. 151.
Suprapubic Prostatectomy. H. M. Ill.—p. 153.
Subarachnoid Hemorrrhage Complicating Sicklemia with Review of Neurological Manifestations and Case Report. M. R. Bronstein. —p. 157.
Raynaud's Disease: Surgical Treatment: Report of Two Cases. G. R. Hardy.—p. 159.
Proctologic Considerations in Intestinal Bleeding. F. S. Forte.—p. 161.
Vitamin E Therapy in Treatment of Myopathies. M. M. Kessler.—p. 164.
Drug Delirium Due to Pyribenzamine: Report of Case with Recovery. I. L. Sperling.—p. 166.

New York State Journal of Medicine, New York

51:1105-1232 (May 1) 1951

500 Patient Years of Rheumatic Disease in Children. R. A. Higgons, G. M. Hyde and M. L. Malsch.—p. 1143.
*Analgesic Action of Sodium Gentisate. R. C. Batterman, L. S. Kryle and S. Dann.—p. 1152.
Postoperative Hepatic Failure with Cholorrhea Following Biliary Tract Surgery. M. N. Friedman and E. K. Mehne.—p. 1155.
*Aureomycin and Penicillin in Treatment of Bacterial Pneumonia: Comparative Study. J. L. Leland and N. Allebach.—p. 1159.
Reversibility of Early Bronchiectasis: Its Implications for Therapy and Prevention. W. Finke.—p. 1163.
Pregnenolone: Preliminary Report on Pregnenolone Acetate and Acetoxypregnenolone in Arthritis and Soft Tissue Rheumatism. W. B. Rawls and V. C. Ancona.—p. 1167.
Treatment of Sciatica with Glucurone. J. H. Hodas, H. Laage and H. Brandon.—p. 1171.
Technic for Measuring Pulmonary Arterial Pressures During Pneumonectomy: Preliminary Report. J. G. Converse, A. Stranahan, A. A. Ehler and M. H. Harmel.—p. 1174.
Adequacy of Prothrombin Synthesis During Terramycin Therapy. R. D. Barnard.—p. 1175.
Cataract Associated with Neurodermatitis Disseminata. J. A. Cetner. —p. 1178.

Analgesic Action of Sodium Gentisate.—Forty-three hospitalized medical and surgical patients and 54 ambulatory patients who had pain varying in causation, type, severity, duration, and organ involved received sodium gentisate. The hospitalized patients received 0.3 and 0.6 gm. of sodium gentisate as a single or repeated dose administered orally several times daily. Most of the patients received eight or more doses. The period of observation ranged from one to 56 days. Satisfactory control of pain was achieved in 34 of the 43 hospitalized patients. None of them showed any sign or symptom of toxicity or an untoward reaction to sodium gentisate. The ambulatory patients were advised to take one or two tablets, equivalent to 0.3 and 0.6 gm., respectively, either as a single dose or in repeated doses. The patients were observed for periods of one day to 30 weeks. Of the 54 ambulatory patients, 31 had satisfactory analgesia with the oral administration of either 0.3 or 0.6 gm. of sodium gentisate. Toxicity occurred in six patients (11.1%), of whom four had nausea, one had pruritus, two dizziness, and one abdominal colic. Thirty-five of the 54 patients were observed under similar circumstances for the effectiveness and toxicity of acetylsalicylic acid (aspirin). Nine of these patients noted no difference in relief of the pain from either aspirin or sodium gentisate. Eleven patients preferred aspirin to sodium gentisate, while the remaining 15 preferred sodium gentisate to aspirin. Of the 35 patients who received aspirin, toxic manifestations were noted in 14 (40%). Sodium gentisate, when administered orally, is a safe and effective analgesic for the relief of mild and moderately severe pain. The analgesic potency of sodium gentisate is equivalent to that of aspirin, but it is more useful because of its lower toxicity.

Treatment of Bacterial Pneumonia.—Of 76 patients with bacterial pneumonia, 36 were treated with penicillin and 40 with aureomycin. Most of the penicillin-treated patients received an initial dose of 500,000 units followed by 250,000 units twice a day until they were afebrile for two to three days. The aureomycin-treated patients received an initial dose of 2 gm. of the drug followed by 1 gm. every six hours until defervescence occurred, then 0.5 gm. every six hours until they were afebrile for three days. There were no significant differences in over-all therapeutic response to the two drugs in the two series of patients. In the penicillin series there were two patients, one with a sterile pleural effusion, the other with active tuberculosis, whose temperatures failed to respond promptly. In the aureomycin series there was one death from Staphylococcus pneumonia in a patient with chronic nephritis and uremia. After two days of aureomycin therapy with no improvement, she was given penicillin and streptomycin, which also were ineffective. There were two other patients whose temperature failed to respond promptly; one had a sterile pleural effusion, and the other had Friedländer A bacillus pneumonia, later cured by streptomycin. Fourteen patients (35%) treated with aureomycin had nausea, vomiting, or diarrhea. There was no toxicity from penicillin.

Ohio State Medical Journal, Columbus

47:297-392 (April) 1951

Therapeutic Team: Consideration of Collaborative Role of Doctor, Nurse, Attendant, and Family. J. L. Fetterman.—p. 321.
Acute Bacterial Endocarditis: Report of Case. J. C. Markley and E. B. Lefken.—p. 326.
Imperforate Anus and Atresia of Rectum: Report of Two Cases Treated by One Stage Abdominalperineal Reconstruction. F. M. Flickinger. —p. 329.
In Situ Carcinoma of Cervix. J. W. W. Epperson and J. M. McCord. —p. 333.
Diagnosis and Treatment of Sterility in Female. J. J. McDonough. —p. 336.
Transverse Presentation Treated by Cesarean Section. J. E. Morgan. —p. 341.

Physiological Reviews, Baltimore

31:107-204 (April) 1951

Intravascular Agglutination of Formed Elements of Blood. B. R. Lutz. —p. 107.
Mucoproteins in Relation to Virus Action. F. M. Burnet.—p. 131.
Fact and Theory Regarding Calorigenic Action of Adrenaline. F. R. Griffith Jr.—p. 151.
The Hepatic Artery. J. Markowitz and A. M. Rappaport.—p. 188.

Public Health Reports, Washington, D. C.

66:547-586 (May 4) 1951

Tuberculosis Mortality in the United States, 1949 (with Provisional Figures for First 11 Months of 1950). E. H. Halpin and O. D. Turner. —p. 547.
Experimental Error in Determination of Tuberculin Sensitivity. S. N. Meyer, A. Hougen and P. Edwards.—p. 561.
Atabrine Therapy of Histoplasma Infections in Mice. C. C. Campbell and S. Saslaw.—p. 570.

66:587-618 (May 11) 1951

Physical Status of Men Examined Through Selective Service in World War II. M. S. Goldstein.—p. 587.

66:619-654 (May 18) 1951

Activities of Medical Administrators in State Health Departments. H. M. Graning, M. Ferguson, B. A. Cheney and A. M. Leflingwell.—p. 619.
Nutrition and Control of Chronic Disease: Public Health Aspects. R. W. Vilter and C. Thompson.—p. 630.
Cultivating Community Relationships in Mental Health Program. H. L. Rooney.—p. 637.
*Influence of Irradiation and Penicillin on Experimental Syphilis Transmission. T. F. Probey.—p. 644.

Transmission of Syphilis by Transfusion.—Laboratory studies in experimental syphilis in rabbits have demonstrated that Treponema pallidum becomes avirulent and will not transmit syphilis when present in blood kept under blood bank conditions for 96 hr. or when in plasma processed to the dried state. Under these conditions blood from donors with syphilis should be acceptable for use. The hazard of transfusion syphilis may, therefore, be minimized by the routine processing of blood or blood products. However, the danger of transmission still exists in emergency transfusions with freshly drawn blood from syphilitic donors. In this event, a sterilizing agent, treponemicidal drug, or possibly irradiation would be required. The studies presented in this paper demonstrated that transmission of experimental syphilis in rabbits by transfusion was not prevented by previous exposure of whole blood containing virulent T. pallidum to ultraviolet irradiation, using the Oppenheimer-Levinson quartz chamber apparatus, or to crystalline peniciilin G in large quantity, added one hour before transfusion.

South Dakota J. Med. & Pharmacy, Sioux Falls

4:77-94 (April) 1951. **Partial Index**

Why Test Visual Fields? P. J. Leinfelder.—p. 77.

4:95-132 (May) 1951

Malignancies of Spinal Cord and Peripheral Nerves. E. Sachs.—p. 95.
Choice of Kidney Incision. R. F. Livingston and W. L. Valk.—p. 100.
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Pathology of New Growth of Thyroid Gland. F. C. Helwig.—p. 109.

Southern Medical Journal, Birmingham, Ala.

44:369-466 (May) 1951

Lipotropic Factors in Treatment of Cirrhosis: Evaluation by Biopsy and by Study of Phospholipid Turnover Employing Radioactive Phosphorus. J. O. Williams, D. Cayer and W. E. Cornatzer.—p. 369.
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Transurethral Resection of Bladder Neck in Treatment of Congenital Abnormalities in Children. J. R. Rinker.—p. 382.
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Surgical Significance of Cardiospianchnic Reflex. D. W. Smith and M. A. Larkin.—p. 399.
Evaluation of Rapid Method of Cytologic Diagnosis in Suspected Skin Cancer. J. M. Hitch, T. B. Wilson and A. Scoggin.—p. 407.
Digitalis in Heart Disease. H. L. Clay Jr.—p. 414.
Effect of Digitalis and Other Drugs on Heart Contractile Force: Clinical Implications. R. P. Walton and P. C. Gazes.—p. 418.
Treatment of Coronary Artery Disease. W. N. Viar and T. R. Harrison. —p. 424.
Office Diagnosis and Treatment of Functional Uterine Bleeding. L. F. Turlington.—p. 429.
Problems of Prostate Gland. G. Carroll.—p. 432.
Treatment of Infantile Bowlegs and Knock Knees with Special Consideration of Therapeutic Use of Large Doses of Activated Ergosterol: Analysis of 58 Cases. H. D. Morris.—p. 435.
Physiologic Labor with Saddle-Block Anesthesia for Delivery. O. A. Ellingson and R. H. Douglas.—p. 439.
Cooperation Between Organized Medicine and Public Health. N. H. Dyer —p. 444.
Chemical Burns of Eye. R. S. McLaughlin.—p. 446.
Practical Therapeutics in Otolaryngology. W. C. Warren Jr.—p. 449.
Effect of Cortisone and ACTH on Bacterial Infections. M. Michael Jr. —p. 450.
Skin Cancer: One-Year Survey in Private Practice. J. G. Thompson. —p. 453.

Surgery, St. Louis

29:643-802 (May) 1951

Acute Pancteatitis and Pancreatic Injuries Following Subtotal Gastrectomy. K. W. Warren.—p. 643.
Partial Esophagectomy with End-to-End Anastomosis. E. B. Kay and K. L. Brown.—p. 658.
*Histopathology of Adenomatous Polyps of Colon and Rectum: With Special Emphasis on Relationship Between Adenoma and Carcinoma. H. E. Bacon, J. Laurens and A. R. Peale.—p. 663.
Pump-Oxygenator to Supplant Heart and Lungs for Brief Periods: I. Evaluation of Oxygenator Techniques; Efficient Oxygenator. K. E. Karlson, C. Dennis, D. Westover and D. Sanderson.—p. 678.
Id.: II. Method Applicable to Dogs. C. Dennis, K. E. Karlson, W. P. Eder and others.—p. 697.
Patent Ductus Arteriosus: Recurrence Following Ligation. C. B. Davis Jr., E. H. Fell and B. J. Gasul.—p. 714.
Growth of Vascular Anastomoses with Continuous Posterior and Interrupted Anterior Silk Sutures. J. Johnson, C. K. Kirby, M. W. Allam and W. Hagan.—p. 721.
Growth of Vena Cava and Aorta Grafts: Experimental Study. J. Johnson, C. K. Kirby, M. W. Allam and W. Hagan.—p. 726.
Melanocarcinoma of Plantar Surface of Foot: Review of 25 Cases. A. M. Decker and J. T. Chamness.—p. 731.
Use of Hyaluronic Acid for Fixation of Skin Grafts. H. H. LeVeen, E. Franklin and J. R. Barberio.—p. 743.
Invasive Infection Due to Genus Pseudomonas. C. W. Howe.—p. 748.
Aureomycin as Adjunct in Treatment of Major Burns. L. T. Wright, J. A. Tamerin, W. I. Metzger and A. L. Garnes.—p. 763.

Adenomatous Polyps of Colon and Rectum.—The origin, pathogenesis and histopathology of adenomatous polyps of the colon and rectum are reviewed. The relation between the benign adenoma and carcinoma is emphasized in this discussion. A pathological study was made of 800 cases of carcinoma of the large bowel. In 162 adenomatous lesions, there were 26 benign adenomas, 16 adenomas with atypia, 61 adenomata malignum or adenocarcinomas in situ, 24 grade 1 adenocarcinomas, five grade 2 adenocarcinomas definitely arising in an adenoma, and 30 polypoid papillary grade 2 adenocarcinomas. Of this series 90 (11.25%) showed definite histological evidence of having arisen in a benign adenomatous polyp, while 30 (3.75%) presented presumptive evidence of such origin. The criteria for the diagnosis of malignancy are presented, and the importance of the character of the epithelial cells is stressed. Close cooperation between the surgeon and the pathologist is urged for a better understanding of the proper management of this important group of neoplasms. Adequate surgical removal of certain polypoid lesions of the colon and rectum should be carried out, even if this requires radical resection in selected cases.

FOREIGN

Annales Paediatrici, Basel

176:297-360 (May) 1951. **Partial Index**

*Insulin Therapy in Chorea Minor. E. Mayerhofer.—p. 297.
Adrenal Neuroblastoma: Report of Four Cases. E. Stransky and N. S. Felix.—p. 308.
Impairment of Liver in Toxicosis. O. Tezner.—p. 321.

Insulin Therapy of Chorea Minor.—In investigating the use of insulin in disorders other than diabetes, Mayerhofer studied muscular disorders, because muscle sugar in the form of glycogen is an important source of energy for the skeletal musculature. Two or three daily injections of from 3 to 7 units of insulin, while a lump of sugar was taken by mouth, together with a diet rich in carbohydrates acted favorably on the muscular unrest in the early phase of chorea. This calming effect appears so quickly, that is, after only a few days, that the author believes that it is not so much the result of local formation and storage of muscular glycogen but that it is rather brought about by a central, probably endocrine improvement in neurological regulation, which facilitates better coordination of muscular movements. It is also possible that both factors act synergistically. Although the efficacy of the insulin treatment in chorea minor cannot yet be fully explained, on the basis of experiences in 21 cases, the author recommends the practical application of the new therapy. It is at least as effective as the older therapeutic methods and, in many although not in all cases, it seems to be the best method. The insulin therapy in chorea is also of a certain theoretic interest. The author discusses muscular and particularly endocrine factors that might play a part in the etiology of chorea.

Beiträge zur klinischen Chirurgie, Munich

182:129-256 (No. 2) 1951. **Partial Index**

History of Local Anesthesia and Its Development into a Method of Treatment. D. Kulenkampff.—p. 129.
Percutaneous Arteriography of Brain and of Extremities with Iodopyracet Injection (Perabrodil®). T. Tiwisina.—p. 142.
*Significance of Parenterally Administered Amino Acids in Preoperative and Postoperative Management. H. H. Westermann.—p. 153.

Treatment with Intravenous Injections of Amino Acids.—Eleven patients with hypoproteinemia were subjected to a therapeutic trial with parenterally administered 10% elamine (purified casein hydrolysate) solution with dextrose, containing a standardized sterile mixture of amino acids. The patients were divided in three groups, one consisting of patients with preoperative and postoperative hypoproteinemia, the second consisting of surgical patients with undisturbed absorption of food in the presence of an unimpaired intestinal tract, and the third consisting of patients with disturbed absorption of food after surgical intervention on the stomach. The administration of amino acids is of greatest importance in patients with chronic suppurative wounds and in patients with disturbed absorption of food. The elamine solution was as a rule administered by infusion of 50 to 60 drops per minute. For prevention of untoward reactions, such as nausea, vomiting, sensation of heat, and headache, the infusion velocity may be reduced. Treatment with intravenously administered amino acids is contraindicated in patients with cardiac decompensation, severe liver insufficiency, or acidosis. Results in the author's patients showed that a positive nitrogen balance was obtained in most of the patients and the protein serum level was restored to normal in all. There was subjective and objective improvement of the general condition. In the course of 50 intravenous infusions of amino acids, 11 of which were given to a single patient, collapse occurred in only one patient with hydatid cysts in the pleura; it was probably due to an antigen-antibody reaction. Intravenous administration of amino acids should be limited to short preoperative treatment and to treatment during the first postoperative days. To prevent a drop in serum protein values after discontinuation of the intravenous therapy, oral or rectal administration of pulverized or dissolved amino acid mixtures is advisable during convalescence. The amino acid mixtures are more effective than blood transfusions in improving the serum protein level in patients with hypoproteinemia. Treatment with blood transfusions on the day of the surgical intervention to combat shock in patients subjected to extensive surgical interventions combined with subsequent intravenous and oral administration of amino acids appears to be most promising.

Brain, London

74:1-114 (March) (Part I) 1951

Surgical Treatment of Colloid Cyst of Third Ventricle: Report Based upon 21 Personal Cases. W. McKissock.—p. 1.
Effects of B. A. L. in Hepatolenticular Degeneration. J. N. Cumings.—p. 10.
Colloid Cysts of Third Ventricle: Analysis of 29 Cases. R. Kelly.—p. 23.
Corticofugal Connexions of Posterior Orbital Surface in Rhesus Monkey. P. D. Wall, P. Glees and J. F. Fulton.—p. 66.
*Nervous and Mental Disorders in Cushing's Syndrome. J. D. Spillane.—p. 72.
Hyaline Bodies in Optic Discs: Report of 10 Cases Exemplifying Importance in Neurological Diagnosis. J. W. Chambers and F. B. Walsh.—p. 95.

Nervous and Mental Disorders in Cushing's Syndrome.—Significant mental and/or neurological disturbances were observed in five of the seven patients with Cushing's syndrome that are reported on in this report. In one patient pituitary irradiation produced apparent clinical cure of the condition, but two years later progressive optic atrophy and cerebral atrophy set in. Four years after treatment the patient was blind, bedridden, and incontinent. Two patients were psychotic in the early stages of the disease. Another patient, who had much emotional instability and was in a neuropsychiatric center for several months, subsequently showed the physical signs of Cushing's syndrome. He also had an attack of unilateral retrobulbar neuritis, which left him with a permanent central scotoma. The fifth patient had considerable depression and alteration of personality in the first year of his illness. In reviewing the literature on the occurrence of nervous and mental disturbances in Cushing's syndrome, Spillane says that many reports do not mention the mental stage of the patient, but he lists 50 reported cases in which there was significant nervous and mental distress. Not included in this list are other cases in which minor symptoms, such as irritability, nervousness, insomnia, forgetfulness, shyness, feelings of self-consciousness and avoidance of social contacts, were the only nervous symptoms mentioned.

British Journal of Dermatology, London

63:165-202 (May) 1951

Tinea Capitis: Review of 1,004 Cases. J. M. Beare and E. A. Cheeseman.—p. 165.
Porokeratosis (Mibelli): Case with Extensive Lesions: Results of Animal Inoculation. J. Savage and H. Lederer.—p. 187.
Treatment of Warts with X-Rays: Is Their Action Physical or Psychological? F. F. Hellier.—p. 193.

British Journal of Industrial Medicine, London

8:53-116 (April) 1951

Role of Periodic Examination in Prevention of Coalworkers' Pneumoconiosis. A. L. Cochrane, C. M. Fletcher, J. C. Gilson and P. Hugh-Jones.—p. 53.
Action of Variable Amounts of Quartz on Lungs of Rats. Extent of Pathological Change in Relation to Amount Injected. S. C. Ray, E. J. King and C. V. Harrison.—p. 62.
*Action of Small Amounts of Quartz and Larger Amounts of Coal and Graphite on Lungs of Rats. S. C. Ray, E. J. King and C. V. Harrison.—p. 68.
Action of Anthracite and Bituminous Coal Dusts Mixed with Quartz on Lungs of Rats. S. C. Ray, E. J. King and C. V. Harrison.—p. 74.
Cardiovascular Disease in Cotton Workers: Part I. R. Schilling and N. Goodman.—p. 77.
Influenza Epidemic in Industry. A. L. Potter.—p. 91.
New Industrial Disease Called Hyperhidrosis Erythematosa Traumatica. J. H. T. Davies.—p. 95.

Action of Quartz plus Coal or Graphite on Lungs of Rats.—It is generally accepted that silica is the cause of silicosis, but it is not known to what degree nonsiliceous dusts assist or retard the silicotic process, when inhaled with silica. The effect of coal dust has been studied before, especially with reference

The items in these letters are contributed by regular correspondents in the various foreign countries.

to its power to increase phagocytosis of siliceous dusts or to absorb the silica released in solution from such dusts. From such studies it had been concluded that inhalation of coal dust should lessen the pathogenicity of siliceous dusts. Many have believed that coal dust protects against silicosis. Others, however, reported cases of acute silicosis from the inhalation of coal dust, and they thought the coal probably hastened the action of the silica. Studies on rats reported in this paper revealed that pure coal dust gives rise to no serious reaction in the animal lungs. Much of it is eliminated, and the amount retained causes only slight reticulinosis. Small doses of quartz that alone produce no lesion in the rats' lungs produce nodular foci of reticulinosis when large amounts of coal dust are added. The large amount of coal probably upsets the drainage mechanism, and the silica retained in the lung exerts its toxic effect with the production of fibrous nodules. Pure graphite was as inert as pure coal, but fibrous nodules were produced when it was mixed with small amounts of silica. It is therefore concluded that the simultaneous presence of large amounts of coal or graphite exaggerated the pathogenic effects of small amounts of silica.

British Journal of Ophthalmology, London

35:189-252 (April) 1951. **Partial Index**

Retinal Micro-Aneurysms in Non-Diabetic Subject. N. Ashton.—p. 189.
Eosinophilic Granuloma of Orbit. A. J. Beller and W. Kornblueth. —p. 220.
Soemmering's Ring and Its Dislocations. G. S. Guha.—p. 226.
Folds in Lens Capsule After Trauma: Sign of Partial Rupture of Zonule. J. L. Bignell.—p. 234.
Adenoma of Limbal Conjunctiva. J. François and M. Rabaey.—p. 237.
Ophthalmomyiasis in England. R. Smith.—p. 242.
Marfan's Syndrome with Retinal Detachment. J. R. Hudson.—p. 244.

British Medical Journal, London

1:1095-1156 (May 19) 1951

Diet in Diabetes. C. C. Forsyth, T. W. G. Kinnear and D. M. Dunlop. —p. 1095.
"Priscol" in Treatment of Peripheral Vascular Disease. J. F. Goodwin and S. Kaplan.—p. 1102.
*Response of Megaloblastic Anaemia of Pregnancy to Crystalline Penicillin G. H. Foy, A. Kondi and A. Hargreaves.—p. 1108.
Clinical Significance of Vitamin-A Deficiency. Z. A. Leitner.—p. 1110.
Distribution of Anopheline Mosquito Bites Among Different Age Groups: New Factor in Malaria Epidemiology. R. C. Muirhead-Thomson. —p. 1114.
Blackwater Fever in African Children. D. B. Jelliffe.—p. 1117.
Laboratory Diagnosis of Urinary-Tract Infections: With Special Reference to Lactose-Fermenting Coliform Strains. J. B. Enticknap and B. J. Stephens.—p. 1119.

Megaloblastic Anemia and Crystalline Penicillin.—Foy and associates have previously described the favorable therapeutic effects of injections of penicillin G in a nonpregnant woman who had megaloblastic anemia and a histamine-fast achlorhydria. Now they report the use of this substance in a woman with megaloblastic anemia who was six months pregnant. She was given injections of 200,000 units of crystalline penicillin G (sodium salt) twice daily for 17 days. On the seventh day of penicillin treatment her reticulocytes rose to 26%, and there was an improvement in the red blood cell count and hemoglobin. When the injections of penicillin were stopped for about 10 days, the blood cell count remained unchanged or fell slightly. Then the injections were resumed, and, 19 days later, when the patient left the hospital, her erythrocyte count was 4,300,000 and the hemoglobin level 12 gm. The response was rapid, complete, and in every way similar to that obtained with liver, folic acid, and vitamin B_{12}. The marrow reverts to normal, the megaloblasts disappearing rapidly and the giant stab cells more slowly. It is suggested that this response to penicillin may be due to any or all of the following: (1) changes in the intestinal flora, which may lead to the destruction of antagonists or competitors, or other factors which may be interfering with the synthesis, absorption, or utilization of hemopoietic factors; (2) interference with bacterial metabolism, so that hemopoietic factors are released for the use of the body; (3) an effect on protein metabolism and the synthesis of nucleoproteins from basic amino acids; (4) the presence in penicillin of hitherto unknown hemopoietic factors, and (5) the destruction of toxins produced by bacteria.

Deutsche medizinische Wochenschrift, Stuttgart

76:661-692 (May 18) 1951. **Partial Index**

Ménière's Disease, Hemicrania and Vascular Innervation in the Region of the Head. P. Beickert.—p. 661.
Differential Diagnosis in Acute Infectious Lymphocytosis. E. Huth and H. Grunze.—p. 662.
*Criticism of Roentgen Diagnosis of Gastritis. A. Müller.—p. 664.
*Sensitization to Factors Other Than Those of the Rh-Group as Cause of Stillbirths: Kell Erythroblastosis. O. Prokop and F. Schleyer. —p. 665.
Experiences with Chediak's Microreaction in Examination of Cerebrospinal Fluid for Syphilis. E. Heinke.—p. 668.

Roentgen Diagnosis of Gastritis.—Müller asserts that gastroscopy frequently shows a normal gastric mucosa after a previous roentgenologic examination had disclosed changes indicative of gastritis. Comparative gastroscopic tests on 5,100 cases revealed that the roentgenologic diagnosis of gastritis was correct in only 61.7% of the cases. In a group of 1,200 cases the incidence of agreement was only 49%. On the basis of such results, the author denies that the roentgenologic demonstration of the relief configuration of the gastric mucosa is of value in the diagnosis of gastritis.

Kell Erythroblastosis.—Serologic investigations of recent years demonstrated that a significant number of formerly unexplained stillbirths could be traced to serologic incompatibility. The most widely known of these are those in the Rhesus system, but in the Lewis system and even in the classical ABO system of blood groups such serologic incompatibilities are also possible. Other factors have been discovered as the result of transfusion reactions, although they have not as yet been known to produce erythroblastosis. In this paper a case is reported in which the so-called Kell antigen was responsible for pregnancy toxicosis and death of a fetus.

Journal de Médecine de Lyon

32:447-510 (May 20) 1951. **Partial Index**

Transcerebral Iontophoresis of Calcium and Secondary Malignant Syndrome of Toxicosis. G. Mouriquand and N. Boulez.—p. 447.
Diffuse Osteopathies in Course of Septicemia in Nursing Infants. M. Bernheim, J. A. Roux and L. Gaillard.—p. 451.
*Brief Considerations on Treatment of 34 Patients with Tuberculous Meningitis in Children's Department of a Hospital, and Results Obtained. P. Bertoye, P. Monnet and J. Thivolet.—p. 457.

Treatment of Tuberculous Meningitis in Children.—Thirty-four children, eight of whom were nursing infants between the ages of 3 and 17 months, with tuberculous meningitis are reported on. Twelve of the 34 patients received streptomycin by the intramuscular route only. Six patients received in addition one course of 15 to 20 intrathecal injections of streptomycin. The remaining sixteen patients received streptomycin intramuscularly and, in addition, intrathecal injections, which were given either daily, to the rapidly deteriorating patients, or in repeated courses of 30 to 45 injections. There were 19 deaths. Recovery resulted in eight patients, seven of whom had been followed up for at least one year after the discontinuation of streptomycin. Six patients are still under treatment, with apparently highly favorable results in four and unsatisfactory results in two. One patient was lost sight of during the fifth month of apparently successful treatment. Only one of the eight nursing infants recovered. Familial contagion was observed in eight children, of whom six died; prognosis is uncertain in the two who survived. For better results with streptomycin therapy in tuberculous meningitis it seems imperative to continue the administration of streptomycin by intramuscular route for more than eight months with either daily injections or intermittent courses and to combine it with intrathecal injections for prolonged periods, until the cerebrospinal fluid has been restored to normal. Repeated courses of 30 to 45 intrathecal injections should be given if necessary. Because of the difficulties of the procedure prolonged hospitalization in a well-equipped hospital is required. Early diagnosis and, consequently, early treatment are of great importance. Diagnosis may be established by the demonstration of Koch's bacillus in the cerebrospinal fluid. Animal inoculation with the cerebrospinal fluid was carried out in 30 of the authors' cases, and positive results were obtained in 26. In the four other cases the diagnosis was established by associated signs and the course of the disease.

Journal Obst. & Gynaec. Brit. Empire, Manchester

58:165-372 (April) 1951. **Partial Index**

Origin of Lower Uterine Segment. W. Shaw and P. Nirula.—p. 165.
Iron Metabolism in Pregnancy: Behaviour of Haemoglobin, Serum Iron, Iron-Binding Capacity of Serum Proteins, Serum Copper and Free Erythrocyte Protoporphyrin in Normal Pregnancy. S. Ventura and A. Klopper.—p. 173.
Resuscitation of the Newborn. L. G. Higgins.—p. 190.
Endocrine Varieties of "Androblastoma" in Ovary and Testis. G. Teilum. —p. 201.
Radiological Pelvimetry and the General Practitioner. W. Hawksworth and E. P. Allen.—p. 203.
*Humoral Origin of Hypertension in Toxaemia of Pregnancy. A. D. T. Govan, C. L. Mukherjee, J. Hewitt and W. F. Harper.—p. 216.
*Chronic Endometritis. J. G. Dumoulin and P. E. Hughesdon.—p. 222.
Prematurity. D. J. Conway.—p. 236.
Acute Rheumatism in Pregnancy. L. I. M. Castleden, J. L. Hamilton-Paterson and E. ap I. Rosser.—p. 253.
Presentation and Prolapse of Umbilical Cord: Analysis of 350 Cases. E. Cope.—p. 259.
Venous Thrombosis During Pregnancy Treated with Dicoumarin. H. P. Wright.—p. 272.
Antenatal Thrombosis. J. L. Hallum and E. M. Newham.—p. 278.
Review of 100 Cases of Transplantation of Ureters in Treatment of Obstetrical Vesicovaginal Fistulae. P. G. Preston.—p. 282.

Hypertension in Toxemia of Pregnancy.—To determine whether all cases of hypertensive toxemia of pregnancy are of humoral origin and to discover whether the serum concentration of gonadotropins bears any relation to such an origin, the authors studied 140 women in the last trimester of pregnancy, all but 20 of whom had hypertension. Studies of serum gonadotropins, urea clearance, water excretion, and blood urea and the tetra-ethyl ammonium bromide reaction were made in the hypertensive patients. Four patients who had essential hypertension had a low serum concentration of gonadotropins, a normal rate of water excretion and did not react to tetra-ethyl ammonium bromide. The others had hypertensive toxemia and formed two groups on the basis of the above tests. The majority had a high serum concentration of gonadotropins, a diminished rate of water excretion, and no reaction to tetra-ethyl ammonium bromide. A small number had a low serum concentration of gonadotropins, a diminished rate of water excretion, and a reaction to tetra-ethyl ammonium bromide comparable to that seen in normal pregnant patients. It is suggested that in most cases of hypertensive toxemia the hypertension is of humoral origin and is related to hyperfunction of the anterior pituitary.

Chronic Endometritis.—At the turn of the century "chronic endometritis" was a convenient diagnostic label for all cases of excessive or painful uterine bleeding not due to neoplasm and a rationale for their treatment by curettage. It was divided into a glandular type with a thick and hyperplastic mucosa, an interstitial type with stromal inflammatory cells, and various combinations and subdivisions. This concept was discredited when the endometrial cycle was described in 1907. At this time "glandular endometritis" was recognized as a normal stage in the development of the premenstrual endometrium. The diagnosis of interstitial endometritis was retained, however, being restricted to the minority of cases showing plasma cells. The authors review the controversy that arose with regard to the importance of plasma cells in the diagnosis of endometritis. Many writers claim that a persisting primary endometritis is never found, either because a large part of the endometrium is shed each month, or, failing this, because the menstrual flow and vertical position of the uterus provide excellent drainage. Only Schröder seems to believe that infection may persist in the basalis and continue to reinfect the functionalis. Among the conditions with which chronic endometritis is associated, tubal infection is held to be the commonest; others include abortion, uterine polyps, sloughing submucous tumors, recent instrumentation, foreign bodies, cervical stenosis, and, doubtfully, chronic cervicitis. Dumoulin and Hughesdon investigated whether one of these conditions is present when some or many endometrial plasma cells are found, and how reliable the finding of plasma cells is as a guide to the presence of adnexitis, if the uterine causes of endometritis can be excluded. The endometria of 1,240 patients were sectioned, and 66 (5.3%) contained plasma cells. Profuse and/or irregular bleeding was the most frequent symptom; less common were pain, discharge, and infertility. Etiological factors in the form of adnexitis, recent pregnancy, or miscellaneous uterine conditions were found in 42 of the 64 cases, and in 30 of the 34 severest cases, i. e., with more than 10 plasma cells per high-power field. In 22 patients no cause could be found, and some of these apparently had an isolated endometritis. The current view that this does not occur is baseless. Severe endometritis, however, probably does not occur alone; its diagnosis is, therefore, of value, and should be used as an indicator of associated disease. As endometrial pathology has not yet caught up with symptomatology, a known gap in our knowledge, such as isolated endometritis, merits further study.

Strahlentherapie, Berlin

84:481-622 (No. 4) 1951. **Partial Index**

Histochemical and Serologic Investigations on Action of Roentgen Rays. T. Hornykiewytsch and G. Seydl.—p. 489.
*Intrauterine Radium Irradiation of Carcinoma of Corpus Uteri at Women's Clinic in Göttingen. H. Czech.—p. 524.
Irradiation of Cancer of Esophagus. E. Günsel.—p. 548.
Small Doses of Roentgen Rays in Irradiation of Inflammatory Processes. H. Strauss.—p. 554.
*Irradiation Therapy in Hypertension. B. Wolf.—p. 559.
Action of Radium Rays on Growing Human Bone. A. L. Meier.—p. 587.
Action Mechanism of Biologic Reactions of Ultrasonics. O. A. Angerer, G. Barth and W. Güttner.—p. 601.

Intrauterine Radium Irradiation of Corpus Carcinoma.—Czech regards the intrauterine introduction of radium as the most important part of irradiation therapy of carcinoma of the body of the uterus, since women in his clinic who received additional percutaneous roentgen irradiation fared no better than those who had received only intrauterine radium therapy. Because it is difficult to ascertain the exact location of the carcinoma within the uterus, triangular, bucket-shaped, and Y-shaped applicators have been used for better spatial distribution of the radium dosage than is possible with a single rod-shaped holder. The author prefers a packing method in which the uterus is packed with a number of small cylindrical holders of nickel-plated brass, with a top that can be screwed on and off and a loop through which a numbered thread is passed. The continuous numbering makes it possible to follow a certain sequence during the introduction and withdrawal of the holders. A platinum tube, 15 cm. in length with a wall-thickness of 0.3 mm. and holding 10 mg. of radium, is placed into each of the brass holders. A uterus of normal size, having a depth of about 8 cm., is packed with about 12 such holders, larger uteri with up to 20 holders. Measurements on wax phantoms revealed the differences in dosage distribution when one rod-shaped radium holder is introduced and when the uterus is packed with the aforementioned small holders. Dosage problems arising from the close proximity of bladder and intestine to the irradiated uterus are also considered.

Irradiation Therapy in Hypertension.—The results obtained in hypertensive patients with sympathectomy induced Wolf to apply roentgen irradiation to the ganglions of the thoracic sympathetic chain. He applied the rays with a focus-skin distance of 30 cm. to a field 10 by 15 cm. corresponding to the fifth to the 12th thoracic vertebrae, giving a surface dose of 40% of a unit skin dose and a focal dose of 20% of a unit skin dose. The technique used was as follows: 200 kv., 15 ma., 1 mm. Cu, half-value layer 1.5 mm. Cu, and unit skin dose 760 r. Most patients were given first a series of three irradiations at intervals of one or two weeks. Depending on the reaction, additional irradiations were given after six to eight weeks. A total of 34 hypertensive patients were treated, and many of them have been kept under observation for two years. The effects of the irradiation differed in the various forms of hypertension. In seven patients, with so-called pale hypertension, improvement was still not evident after a second series of irradiations. In the other patients a wavelike decrease was noticeable after two to four weeks. On the whole, results were satisfactory. In five patients in whom irradiation of thoracic sympathetic ganglions produced no great improvement, irradiation of the region of the diencephalon was also unsuccessful. The favorable results of irradiation of the sympathetic chain were still evident after 22 months in 12 of 20 patients. Improvement was noticeable particularly in the subjective condition of the patients.

Ugeskrift for Laeger, Copenhagen

113:407-442 (April 5) 1951. **Partial Index**

*Pheochromocytoma and Its Diagnosis. Review. J. Riishede.—p. 407.
*Benzodioxan Test in Pheochromocytoma. M. Faber.—p. 413.
Case of Pheochromocytoma in Child. P. Bastrup-Madsen.—p. 420.
Thrombopenic Purpura After Quinidine Sulfate. A. Pinholt Andreasen and R. Christensen.—p. 422.

Diagnosis of Pheochromocytoma. Review.—The increasing number of cases of pheochromocytoma reported during recent years, Riishede says, and the occurrence of two cases in the neurosurgical department of Aarhus Hospital and five cases in nearby hospitals suggest that this usually curable cause of hypertension is not so uncommon as formerly supposed. About 90% of the cases described in the literature developed from the adrenal glands. Endocrine activity seems to be especially pronounced in the tumors localized to the adrenals. The cardinal symptom of the pheochromocytoma is hypertension, which most characteristically is paroxysmal. The disease is thought to pass through the following stages: paroxysmal hypertension alternating with normal blood pressure and increasingly frequent attacks, permanent hypertension with paroxysmal aggravation, and permanent hypertension without characteristic paroxysms. After the intermittent course diagnosis is often impossible. Pharmacologic tests may be helpful in the diagnosis of pheochromocytoma. Of the tests designed to stimulate the discharge of pressor compounds from the tumor and reproduce symptomatic attacks the histamine test is considered the best, but it may be hazardous. The piperoxan (benzodioxan) and dibenamine® (N,N-dibenzyl-beta-chloroethylamine hydrochloride) tests are designed to lower the blood pressure by competitive inhibition of the pressor compounds secreted by the tumor. Abdominal exploration may be necessary. The only method of treatment of pheochromocytoma is operative removal.

Piperoxan Test in Pheochromocytoma.—Faber recommends the piperoxan (benzodioxan) test as a reasonably reliable test for the presence of pheochromocytoma with persisting hypertension. The test is advised first and foremost in all cases of hypertension with symptoms of paroxysmal attacks, also in cases in which the family history gives no information on the occurrence of hypertension. Some form of pharmacologic pheochromocytoma test is advised in every young hypertensive patient without demonstrable renal affection, as a positive outcome in most cases indicates that curative treatment is possible. Pheochromocytoma appears to occur most commonly between the ages of 20 and 50, but cases with symptoms have been reported in patients aged up to 72. Two cases of operatively verified pheochromocytoma are described, in one of which the piperoxan test was negative. Explanations for the negative outcome are suggested.

113:443-472 (April 12) 1951

*Intoxication on Therapeutic Application of Acetylsalicylic Acid in Infants and Small Children with Colds or Pneumonia. T. Iversen and E. Schiødt.—p. 443.
Fibrous Dysplasia of Bones. K. Lundbaek and R. Olsen.—p. 455.

Intoxication on Therapeutic Application of Aspirin in Infants and Young Children.—Iversen and Schiødt discuss the symptoms of acetylsalicylic acid (aspirin) poisoning in young children on the basis of 39 cases from the literature and their four personal cases, in two of which bronchopneumonia was complicated with acetylsalicylic acid intoxication. The main symptom, they find, is hyperpnea. The patient is usually dehydrated, may be apathetic and delirious, and in grave cases coma and shock dominate. There are changes in the acid-base balance, and the electrocardiogram shows prolongation of ventricular systole. In differential diagnosis, pneumonia, myocarditis, sepsis, and diseases causing acidosis are mainly to be considered. Treatment consists of the administration of 0.9% sodium chloride and 5% glucose; 1.3% sodium biocarbonate should always be given, subcutaneously or intramuscularly, but in small doses, during the first stage of the intoxication. The authors assert that acetylsalicylic acid should not be given in the first years of life, cautioning particularly against its application in suspected or proved pneumonia.

Wiener klinische Wochenschrift, Vienna

63:301-320 (April 27) 1951. **Partial Index**

Functional Improper Posture with Secondary Myalgia and Improper Posture Resulting from Myalgia. A. Saxl.—p. 301.
Goiter in Young Persons. H. Steiner.—p. 303.
*Experiences with Treatment of Hemolytic Disease of Newborn Infants by Exchange Transfusions or Blood Transfusions. H. Kölbl.—p. 307.

Treatment of Hemolytic Disease of Newborn Infants.—Of 34 newborn infants, 25 boys and nine girls, with severe jaundice, 19 were treated by repeated blood transfusions and 15 by exchange transfusions. Rh incompatibility was present in 32 infants, and A-B-O isoimmunization in two. Transfusions are indicated when the erythrocyte counts reveal a rapid drop below 3,000,000. Rapid deterioration of the clinical picture, the occurrence of cutaneous hemorrhage as a sign of capillary damage, the early occurrence of severe jaundice within the first 12 hr. after delivery and a positive reaction to Coombs' test are decisive criteria for the administration of exchange transfusions. The number of the erythroblasts in the peripheral blood is not decisive. Wiener's cumbersome technique of exchange transfusion was used in only one instance, and the Diamond technique or the modified Gasser technique were preferred by the author. In cases in which the umbilical vein was thrombosed, a polyvinyl catheter was introduced into the saphenous vein and pushed forward to the inferior vena cava, thus facilitating a rapid exchange of blood. About 500 to 700 cc. of blood is required for the largest possible exchange of the blocked erythrocytes of the infant. The author cautions against the use of Rh positive blood for exchange transfusions, since this may produce increased hemolysis, increased bilirubinemia, lower nephron nephrosis, and a rapid and enormous increase of the nonprotein nitrogen values, resulting eventually in death. Thirteen of the 34 infants had kernicterus; four of them were treated by exchange transfusion and nine by repeated blood transfusion. Six of the 13 died. The transfusion therapy has no effect on the kernicterus, which is present at birth. Transfusion treatment is not lifesaving, since danger to life depends on the kernicterus rather than on the anemia. Rh or A-B-O incompatability between mother and infant is not a contraindication to breast feeding.

63:357-372 (May 18) 1951. **Partial Index**

*Fall of Circulating Eosinophils Following Administration of Epinephrine. H. Braunsteiner, F. Pakesch and H. Vetter.—p. 359.
Antihistamine Preparation Used as Prophylactic in Burns. T. Wense.—p. 362.
Nailing of Pertrochanteric Fractures of Femur. G. H. Marcus.—p. 363.

Fall of Circulating Eosinophils After Administration of Epinephrine.—The fall of circulating eosinophils after the intramuscular injection of 0.3 mg. of epinephrine and the usefulness of this phenomenon as a test for adrenal cortex deficiency was studied in 114 persons. This group included eight patients with a clinical diagnosis of adrenal cortex deficiency, 60 persons who were normal or slightly ill without clinical signs of adrenal cortex deficiency, 20 patients with moderately severe disease, and 26 who were chronically ill. Initial eosinophil counts varied from less than 80 to more than 4,000 cells per cubic millimeter. Results showed that the absence of a 30% or more fall in circulating eosinophils within four hours after the administration of epinephrine on repeated examination presents a highly probable but not infallible indication for adrenal cortex deficiency. The reliability of the test may be impaired by extremely low or high initial eosinophil counts and by chronic diseases. Six of the eight patients with a clinical diagnosis of adrenal cortex deficiency had Addison's disease, for which they had not been treated, and five of these showed a fall in eosinophils of less than 40%. Results of tests of patients with clinically latent or mild adrenal cortex deficiency were not conclusive. Additional experiments showed that *l*-nor-epinephrine has no eosinopenic action, while ephedrine induces a more than 50% fall of eosinophils. The fall of eosinophils after the administration of epinephrine could not be inhibited significantly by sympathicolytic drugs such as N,N-dibenzyl-beta-chloroethylamine (dibenamine®) and dihydroergotamine, or by barbituric acid derivatives, massive doses of desoxycorticosterone or progesterone.

BOOK REVIEWS

Toxicology of Uranium: Survey and Collected Papers. Edited by Albert Tannenbaum, M.D., Director, Department of Cancer Research, Medical Research Institute, Michael Reese Hospital, Chicago. National Nuclear Energy Series, Manhattan Project Technical Section, Division IV—Plutonium Project Record, Volume 23. Cloth. $3. Pp. 333, with illustrations. McGraw-Hill Book Company, Inc., 330 W. 42d St., New York 18; Aldwych House, Aldwych, London, W.C.2, 1951.

A continuation of the long series planned by the United States Atomic Energy Commission, this volume contains reports of animal experiments conducted between 1942 and 1946 on the effects of ingestion and injection of uranium compounds. This work was undertaken because of the need of protecting the many technical workers engaged in the development and production of atomic bombs.

The book is very well written, has a good index, and in general is so designed that it will be exceedingly useful to anyone doing research in the future in this particular field. Material of special interest abounds; an example is the description (pages 274 to 282) of a microfluorometric method capable of determining quantitatively as little as 0.0001 microgram of uranium. The book is naturally not of immediate interest in the practice of medicine but deserves commendation as a most substantial contribution to medical science.

Lehrbuch der Röntgendagnostik. 2.-4. Lieferungen: Skelett. Von H. R. Schinz, W. E. Baensch, E. Friedl und E. Uehlinger. Nebst Beiträgen von E. Brandenberger, et al. Fifth edition. Paper. 70 marks; 82 marks; 84 marks. Pp. 427-878, with 764 illustrations; 879-1406, with 732 illustrations; 1407-1920, with 674 illustrations. Georg Thieme Verlag, Diemershaldenstrasse 47, (14a) Stuttgart O; agents for U. S. A.: Grune & Stratton, Inc., 381 Fourth Ave., New York 16, 1950; 1951.

The review of Section 1 appeared in THE JOURNAL Nov. 4, 1950. Sections 2, 3, and 4 are concerned with the skeleton.

Section 2 deals with aseptic necrosis, toxic lesions of bone, inflammatory diseases of bone, certain of the hereditary skeletal defects, and some of the primary neoplasms. One may argue with the validity of listing Paget's disease and Caffey's disease as "inflammatory," but a completely logical classification does not exist, unless one employs a "catch-all" group of diseases of "unknown etiology."

The first 53 pages of Section 3 continue and conclude the consideration of primary malignant neoplasms and are followed by 35 pages on skeletal metastases. The discussion of skeletal anomalies begins with a consideration of the roles played by genetics and by disturbances of physiology of the developing fetus, followed by the generalized anomalies of twins and then of single fetuses. The portion on hypervitaminosis and hypovitaminosis is noteworthy, particularly for the illustrations of severe rickets involving extremities and the sternal ends of the ribs—the so-called rachitic rosary.

The section on disturbances of growth and maturation deals with the various types of dwarfism and gigantism, and under the caption "Hormonal Diseases of Bone" the authors discuss the skeletal results of disturbances of the pituitary, parathyroids, adrenals, ovaries, testes, thyroid, and pancreas. Included, also, are certain forms of osteoporosis, osteosclerosis, and fibrous dysplasia suspected of having a hormonal basis.

The section devoted to joint diseases begins with a protracted discussion of a procedure that has met with little favor in the United States, namely, the direct demonstration of the capsule by injection of positive or negative contrast media—so-called arteriography. Dislocation with and without fracture are well illustrated, and there are included here certain types of fractures that occur in epileptics and in patients subjected to shock therapy.

Arthritis is classified and described in the conventional manner but with the addition of several of the rarely encountered special forms of chronic arthritis. Thirty pages are devoted to hereditary lesions of joints, and here, surprisingly, one finds gout.

Section 4 is devoted to the various diseases of the spine, the skull, muscles, tendons, subcutaneous tissue, blood vessels, breast and the salivary glands, and the localization of foreign bodies. Finally, there are 60 pages of charts, tables, legends, and sketches epitomizing the differential diagnosis of skeletal lesions.

It is only natural that in all three sections the authors have drawn largely on the rich German literature for their text and illustrations, but when they do refer to American work their references usually are to textbooks and secondary publications rather than to basic sources in experimental medicine, surgery, and pathology. Presumably an index will be supplied when all sections of the 5th edition have been published, but the individual sections would have been even more useful than they are at present if each could have had its own separate index, pending the appearance of the cumulative index.

The excellence foreshadowed in Section 1 is continued in sections 2, 3, and 4 and presumably will continue in the numerous sections to follow. These books are a source of complete, authentic, and well illustrated information on specific problems in x-ray diagnosis.

The Enzymes: Chemistry and Mechanism of Action. Volume I, Parts 1 and 2. Edited by James B. Sumner and Karl Myrbäck. Cloth. $13.50; $12.80. Pp. 724; 725-1361, with illustrations. Academic Press, Inc., 125 E. 23rd St., New York 10, 1950; 1951.

Many advances have been made in enzymology in the last several years, and the problems now encountered are of interest to scientists in various fields. Although enzymes originally may have been of interest principally to the biochemist, they have become the subject of study in all the biological sciences as well as the fields of organic and physical chemistry.

In order to evaluate properly the progress made and at the same time point out the many problems still to be investigated, it was desirable to bring together in a comprehensive survey the work that has been done in the enzyme field. This was the thought in mind in the planning of this two-volume series on enzymes, which will be published in four parts. The various aspects of enzymology will be covered by some 87 authorities from Australia, Europe, and the United States.

Part 1 of Volume I contains an introductory chapter by the co-editors of this series. It contains several generalities concerning enzymes, such as their chemical nature, general properties, and the determination of their activity, as well as the definitions, terminology, and classification commonly used in enzyme chemistry. Chapters 2 through 8 deal with certain chemical or physical aspects of enzymes which may be considered to be held more or less in common by all of them. Kinetics, specificity, adaptation, inhibition, and also the role enzymes play in immunochemistry and chemotherapy and their relation to cell chemistry, viruses, hormones, and vitamins are discussed. Chapters 9 through 19 include specific enzymes or groups of enzymes. Among these are the fat and ester hydrolyzing enzymes, cholinesterase and acetylcholinesterase, phosphatases, sulfatases, alpha-D-glucosidases, and beta-glucosidases and other sugar-splitting enzymes, amylases, beta-glucuronidase and invertase.

Part 2 of Volume I is a continuation of the study of the various specific enzymes or enzyme groups. It contains 24 chapters dealing with enzyme groups, such as the cellulases, desulfhydrases, mucopolysaccharases, phosphorylases, transaminases, and transmethylases, and the specific enzymes: carbonic anhydrase, aspartase, fumarase, aconitase, enolase, thiaminase, penicillinase, histidase, urocanase, hippuricase, allantoinase, allantoicase, arginase, and urease. Other class enzymes discussed are the pectic, proteolytic, and nucleolytic enzymes. The role of enzymes in blood and milk coagulation as well as their presence in bacterial and animal toxins are also included.

Since each chapter is prepared by a different authority, the organization of the chapters and the material discussed at length differ considerably. Overlapping and repetition, which might be expected in a book prepared by 87 authors, appear to be minimal.

The reviews here published have been prepared by competent authorities and do not represent the opinions of any official bodies unless specifically stated.

Each chapter has appropriate references, including material as recent as 1950. It has been impossible to include in detail everything that is available; however, some procedures are included where they appear necessary. Part 2 contains the author and subject indexes for both parts of the volume. The series can almost be considered a must for workers or prospective workers in enzymology, whether they are in the medical field, in organic or physical chemistry, biochemistry, zoology, bacteriology, or in any of the other divisions of the biological sciences.

Climate in Everyday Life. By C. E. P. Brooks, I.S.O., D.Sc., F.R.Met. Soc. Cloth. $4.75. Pp. 314, with 34 illustrations. Philosophical Library, Inc, 15 E. 40th St., New York 16, 1951.

From the rich and numerous but widely scattered sources of information on climatology, Brooks first assembled facts for a lecture on climate and the deterioration of materials; this grew into a book on industrial climatology, and finally became the present even more inclusive volume. Its three major divisions are headed "Living with the Climate," "Climate as an Enemy," and "The Control of Climate." The third is of particular medical interest because it includes discussions of heating, air conditioning, lighting, and clothing. Readers curious about the new word "clo" will find a definition on page 254. The surprising integrations of modern life are exemplified on page 252, where the author notes the increased drain on the electric generator systems when a dense thundercloud darkens the sky over a great city. The author's discussion of lighting is far from complete, and in general it becomes evident that the subject of climatology, even in its purely medical aspects, has grown to the point where it can no longer be covered in detail in a volume of this size. Nevertheless, the author deserves credit for having produced a work that is replete with both information and inspiration.

Essentials of Nutrition. By Henry C. Sherman and Caroline Sherman Lanford. Third edition. Cloth. $4.25. Pp. 454, with 36 illustrations. The Macmillan Company, 60 5th Ave., New York 11, 1951.

The newest edition of this popular textbook has been brought up to date and revised in the light of our rapidly expanding knowledge of the science of nutrition. All the data on the nutrient content of foods have been revised to conform to the governmental publication of June, 1950. The authors have not merely added new sections to the different chapters but have completely rewritten them, incorporating the results of recent nutritional research. The bibliography at the end of each chapter has been expanded to include references to basic nutritional research performed since the previous 1943 edition. There is a short discussion of the newer specific factors for calculating the caloric values of foods, and a table in the appendix gives a comparison of these values with those arrived at by the commonly used Atwater factors. Examples of the long-term benefits to be derived from the liberal intake of calcium and vitamin C are also cited. As the authors say in the preface, ". . . today's essentials of nutrition deal with potentialities beyond those that the science of yesterday thought possible."

This book can be highly recommended for use in the teaching of nutrition courses in colleges and nursing schools, and any person who desires a brief comprehensive survey of the field of nutrition will find it of particular value.

Medicinal Chemistry. Volume (E.S.) I. C. M. Suter, Editor-in-Chief. Series of reviews prepared under auspices of Division of Medicinal Chemistry of American Chemical Society. Cloth. $12. Pp. 473. John Wiley & Sons, Inc., 440 4th Ave., New York 16; Chapman & Hall, Ltd., 37-39 Essex St., Strand, London, W.C.2, 1951.

This is the first of a series of volumes sponsored by the Division of Medicinal Chemistry of the American Chemical Society. The stated purpose of the series is "to summarize the available data on the biological properties of medicinal substances and to correlate the relationship between chemical structure and physiological activity in each area covered."

The areas covered in this volume are antithyroid compounds, antispasmodics, antibiotics from plants, benzoates and substituted benzoates as local anesthetics, and analgesics divided into the aralkylamines and partial structures related to morphine. The same general plan is followed for each topic: lists of all compounds that have been tested for a given pharmacological activity, relationship between chemical structure and pharmacological action, brief discussion of methods of synthesis and pharmacological test methods, and inclusion of an extensive bibliography, including references to patents.

There are a great number of tables comparing the activities of numerous compounds organized according to chemical structure. The authors are industrial research workers active in the fields about which they write. The work meets a great need among chemists and pharmacologists who synthesize and evaluate drugs.

Pain and Its Problems. Edited by Sir Heneage Ogilvie, K.B.E., D.M., M.CH., and William A. R. Thomson, M.D. Practitioner Handbooks. Cloth. $3. Pp. 194. The Blakiston Company (Division of Doubleday & Company, Inc.), 1012 Walnut St., Philadelphia 5, 1950.

This is a compilation by various authors of their original short review articles which appeared in *The Practitioner*, plus certain additional chapters. It is by far the best available small monograph on clinical pain. Certain subjects, such as headache, are sketchily disposed of, but the treatment of other types of clinical pain is more than adequate. The two chapters on the drug treatment of pain can be criticized for the use of such typical British drug names as pethidine (meperidine) and omnopon. A few explanatory footnotes might have made the American edition more useful for those practitioners who are unfamiliar with British drug names.

Patterns of Sexual Behavior. By Clellan S. Ford, Ph.D., Associate Professor of Anthropology, Yale University, New Haven, and Frank A. Beach, Ph.D., Professor of Psychology, Yale University, New Haven. With foreword by Robert Latou Dickinson, M.D. Cloth. $4.50. Pp. 307, with 16 illustrations. Paul B. Hoeber, Inc. (Medical Book Department of Harper & Brothers), 49 E. 33d St., New York 16, 1951.

Development of serious interest in the problem of sex education and how it may be carried out most successfully make this volume especially desirable, because all too often those attempting to provide such education have little or no knowledge regarding the evolution of sex behavior in man. The authors have made a survey that includes American society, 190 other peoples, and several groups of animals in the same subclass of mammals. The recognition that there is an extremely wide variation in attitudes regarding sexual behavior could conceivably have a salutary effect on the way in which some sex educators approach their task. The nature of coitus, sexual attractiveness and stimulation, homosexual behavior, and physiological factors in sex behavior are developed in 12 chapters, with a final summary chapter. A glossary and an extensive bibliography are also included.

Advances in Enzymology and Related Subjects of Biochemistry. Volume XI. Edited by F. F. Nord. Cloth. $9. Pp. 471, with illustrations. Interscience Publishers, Inc., 250 5th Ave., New York 1; Interscience Publishers, Ltd., 2a Southampton Row, London, W.C.1, 1951.

As in the other volumes in this series, there are reviews by experts of recent advances in various fields of biochemistry related to enzymes. Thus there are chapters on the biochemical role of entropy by Gutfreund, on reactions at interfaces (including information on toxicity of ions, oxidation of sterols, and action of snake venoms on surface films) by Danielli and Davies, on chlorophyll by Wassink, on pectic enzymes by Lineweaver and Jansen, on transformations of starch by Peat, on alliin (including a short discussion on possible use in therapy) by Stoll and Seebeck, and on wilting in plants by Gäumann. Of most interest to medical scientists, though, are the chapters on the thiol groups of biological importance by Barron of the Chemical Division, Department of Medicine, University of Chicago, and on enzymic synthesis of polysaccharides by Hehre of the department of bacteriology and immunology, Cornell University Medical College. The indexes to this volume are supplemented by a cumulative author index to the series. The printing and binding are excellent.

QUERIES AND MINOR NOTES

EFFECTS OF SECONDARY X-RADIATION

To the Editor:—*I perform on the average of four retrograde pyelograms a week. I use a standard urological table, stand at the foot of the table to inject the contrast media, and remain there during a second and sometimes a third exposure. The tube is a CRT 1-2 Coolidge X-ray Tube Unit. My questions concern the effects of secondary x-radiation that one would be exposed to in the use of this particular tube. Is there a possibility of radiation sterility or other detrimental x-radiation effects? There is no cone or filter on our tube, and we use the small focal spot.*

M.D., Florida.

Answer.—The dosage rate of primary and scattered radiation received by the operator depends on the kilovoltage, milliamperage, and time factors employed, as well as the individual output characteristics of his particular x-ray tube in its housing and the location in which it is used. It also depends on the distance the operator maintains from the x-ray tube, the patient, and the cassette during the exposure. The dosage rate the operator receives should therefore be checked by an instrument that measures ionizing radiations. Such a service can undoubtedly be obtained from the manufacturer of the equipment the operator uses; it is suggested that he communicate with the company's representative or arrange for some qualified person in his locality to measure the radiation in question. The possibility of detrimental effects can only be evaluated after the dosage and an estimate of the quality of the radiation are known. The problems should not be neglected.

It is also suggested that a suitable cone and filter be employed for the protection of both the patient and the operator against unnecessary exposure to ionizing radiations and so that better roentgenographic definition may be obtained.

INFECTIOUS HEPATITIS AND "SPLIT EYE"

To the Editor:—*Is there any relation between infectious hepatitis in humans and "split eye" in poultry, characterized by enlarged liver and transmitted by the droppings from one chicken to another? Infectious hepatitis recently developed in two elderly patients; they eat three to four dozen eggs a week from their own flock. A case of "split eye" was detected in their flock two months before the first case. The second case developed almost exactly 60 days after the first one.*

Arnold C. Tait, M.D., Sunnyside, Wash.

Answer.—Infectious hepatitis of human beings appears unrelated to the various expressions of the avian leukosis complex (ALC), of which ocular lymphomatosis, also occasionally called "split eye," is considered a part. While both diseases are attributed to virus agents, the properties of each agent and disease are distinctive and divergent. Ocular lymphomatosis is characterized by mononuclear cell infiltration, which is apparent chiefly in the iris. This expression of the ALC may be accompanied by hepatolymphomatosis, which, in turn, may be manifested by slight to massive lymphomatosis of the involved organ and is similarly due to an oncogenic agent. Concentric annular or spotty depigmentation or diffuse bluish gray fading of the iris, in one or both eyes, commonly results. The pupil may become irregular with angular indentations ("split eye") and gradual loss of light accommodation. The iris, in advanced cases, is diffusely gray with pinpoint pupil. Slightly turbid exudate collecting in the anterior chamber tends to increase the convexity of the cornea as in glaucoma.

Havens and Paul (in Rivers, Viral and Rickettsial Infections of Man, Philadelphia, J. B. Lippincott Company, 1948) emphasize the characteristic diffuse involvement of the liver in infectious hepatitis in man. There is early and diffuse parenchymal destruction of this organ in contrast to the chronic, largely noninflammatory cellular infiltration that typifies the classic ocular lymphomatosis of domestic fowl and other birds.

HORMONAL BALANCE OF PREGNANCY AND THE MENOPAUSE

To the Editor:—*In women, either during pregnancy or the menopause, occasionally bright red capillary nevi, 1 or 2 mm. in size, develop on the chest, breast, and upper abdomen, sometimes accompanied by moist red palms and painful, fissured pale tongue. What hormonal imbalance, if any, do these women have in common? Is there any connection between protein and liver metabolism, vitamin B deficiency, low estrogen, and high circulating levels of follicle-stimulating hormone? These are not the usual "spider nevi," and none of these women have cirrhosis or alcoholism.*

M.D., Massachusetts.

Answer.—The hormonal balance of pregnancy differs greatly from that of the menopause; in the latter there is a deficiency of estrogen. During pregnancy large amounts of estrogens circulate in the blood and are present in the tissues of the body; in addition, there are unusually large amounts of progesterone as indicated by pregnandiol excretion as well as the high titers of chorionic gonadotropin, which are the basis for many of the pregnancy tests depending on ovarian stimulation in animals. It is therefore highly doubtful that the capillary changes and the fissured tongue have a common endocrine etiology. However, in a deficiency of estrogen, such as occurs in the menopause, capillaries are more fragile. Fissuring of the tongue has little or no relation to estrogen deficiency, although it is known that the mucosa of the mouth and nose may undergo a degree of atrophy in the presence of estrogen deficiency. Whether this may be reflected in changes in the tongue epithelium is not well established. There is little physiological basis for an explanation of such changes during pregnancy. There are typical epithelial changes during pregnancy, such as hypertrophy of the gums, herpes, and petechia, but it is difficult to correlate these changes and those mentioned in the question.

There is a definite relation between liver metabolism and the estrogens. When the liver functions poorly because of organic lesions, protein deficiency, or a vitamin B deficiency estrogens are not destroyed normally, with a resultant increase in circulating estrogenic hormones. This should result in a low level of follicle-stimulating secretion by the anterior pituitary. In long-standing liver diseases, capillary disturbances may appear, and these are possibly due to excessive estrogen over a long period of time. It is doubtful that this situation occurs during pregnancy because of the elevated titers of estrogen.

ASTIGMATIC MICROSCOPISTS

To the Editor:—*For a person unable to wear glasses when working with the microscope, do you know of any arrangement for getting around this difficulty, such as having lenses made from the oculist's prescription fitted over the eye pieces?*

M.D., Michigan.

Answer.—Ordinarily a person working with a single eye piece microscope can compensate enough for the absence of glasses by adjusting the microscope. When substantial astigmatism is present, or if there is a sharp difference in visual acuity based on refractive errors, and a binocular microscope is used, correction usually can be made by adjusting each focussing eye piece separately. To provide for astigmatism of a high degree, it is possible to have the astigmatic portion only of the person's glasses ground into a small lens, which is mounted in a rubber gasket and slipped over the eye piece and set in a definite axis. The latter correction may be monocular or binocular.

The answers here published have been prepared by competent authorities. They do not, however, represent the opinions of any official bodies unless specifically stated in the reply. Anonymous communications and queries on postal cards cannot be answered. Every letter must contain the writer's name and address, but these will be omitted on request.

ASCARIASIS

TO THE EDITOR:—*I have several young patients who began passing adult ascarides after oral penicillin therapy for various illnesses. There are no symptoms referable to the infestation, but three of the children still show fertilized ova in repeated stool examinations. I would appreciate suggestions.*

Harold A. Wente, M.D., Rochester, Minn.

ANSWER.—Hexylresorcinol in hard gelatine capsules (crystoids® anthelmintic) is the drug of choice. This drug is less toxic and more efficient than the others. The capsules must be swallowed without chewing, for the chemical is irritating to the buccal mucosa. Even young children can be taught to swallow pills by practicing first with placebos. The drug is taken in the morning on an empty stomach. No preliminary purge is needed. Food is withheld for five hours. Two hours after administration of the drug a saline purge is given to rid the patient of dead and dying worms. The dose recommended for adults is 1 Gm.; the dose for preschool children is 0.6 Gm., and for children from 6 to 10 years of age the dose is 0.8 Gm.

Hexylresorcinol yields about a 90% cure. It does not kill the larvae outside the intestinal tract, so that treatment may have to be repeated in one week. Reinfection, of course, should be avoided. The tablets must not be too old, for in such cases the hexylresorcinol tablets may not dissolve high enough in the bowel to be effective.

Milk of higueron from Central America is an efficient drug in elimination of ascarides. Its action is that of a proteolytic enzyme which is extracted from the fresh sap of Ficus glabrata and doliario.

BALATA AS AN ALLERGEN

TO THE EDITOR:—*Have you had any reports of epidermal or respiratory symptoms in persons who have been working with webbing impregnated with balata?*

Clifford H. Kalb, M.D., Milwaukee, Wis.

ANSWER.—On a precise basis balata is the sap or dried gum of Mimusops globosa, a tropical or subtropical tree. Less precisely, the term is applied to a variety of vegetable gums, including at times chicle or zapota. Millions of golf balls have had casings of balata without known harm to golfers. In this and similar manufacturing operations, the balata requires solution such as in benzene, carbon disulfide and kerosene. Clearly, exposures to these several solvents could lead to dermatitis or systemic damage. Balata is credited, by Schwartz and Tulipan, with a capacity to provoke dermatitis. The balata of golf balls and presumably that appearing on impregnated textiles is well polymerized and freed of solvent after full drying, but balata as introduced into machinery belting dressings may exist in a more liquid state. The nature of the "webbing" mentioned in the query is not stated. If this represents jute, it may be recognized that this vegetable matter may induce both dermatitis and bronchitis from its dust. It is suspected that balata, if incompletely polymerized, may constitute an allergen just as the highly useful gum acacia is a known allergen. Judiciously applied patch tests might establish sensitivity to balata or to the unimpregnated fabric.

BLOOD AND SPINAL FLUID SEROLOGIC TESTS

TO THE EDITOR:—*When the blood Wassermann test for syphilis is positive, is the spinal Wassermann test always positive and vice versa, in the same case? In case they do not always agree, in what ratio are they always the same?*

W. C. Hensyl, M.D., Berwick, Pa.

ANSWER.—Patients with positive serologic reactions for syphilis do not necessarily have positive spinal fluid Wassermann reactions, unless there is a syphilitic involvement of the central nervous system. Furthermore, most patients with syphilis of the central nervous system will present positive serologic reactions in the serum. Only on rare occasions will a patient have neurosyphilis with positive serologic reactions of the spinal fluid and negative serologic reactions of the blood.

SIMMONDS' DISEASE

TO THE EDITOR:—*A 24 year old, single woman office employee has nausea, vomiting, weakness, loss of appetite, insomnia, pain in the right lower abdominal quadrant and gradual loss of weight from 120 to 82 pounds (54 to 37 Kg.) in the past year. Her hair is graying, and her scalp and skin are dry. Past history includes appendectomy in 1946, exploratory operation for cystic ovary again in 1946 (ovaries are intact) and exploratory operation for adhesions in 1948. She has had no serious illnesses. Menarche occurred at age of 13. The menses were regular, every 28 days, until the appendectomy in 1946, after which periods occurred every three to six or seven weeks. Last menstrual period was December 1949. Her height is 62 inches (157 cm.) blood pressure 82/56 and basal metabolic rate — 28 per cent. Barium examination of the colon disclosed no abnormalities. The kidneys are normal in size, shape and position. Roentgen examination of the skull revealed a normal sella turcica, no evidence of erosion or bone distruction about the sella; bones of the vault are normal, and there is no evidence of chronic pressure. Results of pelvic and rectal examinations were completely negative. Red blood cells numbered 3,800,000, and hemoglobin was 76 per cent. Is this sufficient for a diagnosis of Simmonds' disease? Please outline treatment.*

Stephen R. Minor, M.D., Worcester, Mass.

ANSWER.—The data presented are not sufficient to permit a diagnosis of Simmonds' disease. One would expect atrophy of the breasts, absent pubic and axillary hair and atrophy of the vaginal mucosa. In addition to the basal metabolic study, laboratory studies measuring the function of at least two other glands are necessary: (1) function of the pituitary to be determined by the excretion of the follicle-stimulating hormone in the urine, the insulin tolerance test and the response of the eosinophils to epinephrine and (2) function of the adrenal cortex to be determined by the excretion of the 17-ketosteroids and of the 11-oxycorticosteroids in the urine, the level of sodium, potassium and chloride in the blood and the Cutler-Power-Wilder test. Studies of the ovarian function may also yield valuable information, namely, vaginal smear, endometrial biopsy and excretion of pregnandiol in the urine.

The treatment of Simmonds' disease resolves itself into the treatment of thyroid hypofunction, plus the associated hypogonadism and hypoadrenocorticism. Thyroid by mouth, starting with 30 mg. and increasing slowly to 60 mg. should be administered. Extra salt by mouth, as well as 4 to 6 Gm. of salt in tablet form, should be kept up indefinitely. Small doses of testosterone 10 to 25 mg., given intramuscularly twice a week, help protect against adrenocortical failure in either sex. In females moderate doses of estrogens may replace testosterone. In young patients chorionic gonadotropin, in doses of 500 to 1,000 units intramuscularly twice a week will stimulate the gonads satisfactorily. Cortisone or pituitary adrenocorticotropic hormone (ACTH) may be used to improve the adrencortical insufficiency in this disease. Small doses (25 to 50 mg. daily) may be given over a prolonged period of time.

PTYALISM DURING PREGNANCY

TO THE EDITOR:—*What is the treatment of ptyalism associated with pregnancy? A 32 year old woman, gravida II, experienced ptyalism during her first pregnancy. She is now in her second month and, due to excessive salivation, is having discomfort. Tincture of belladonna and pyribenzamine® have been employed with no marked relief. For a short time the latter appeared to be of some aid, but, because of the side effects and the pregnancy, it was discontinued. Are there any nontoxic drugs that are effective in controlling the ptyalism?*

Herman Rice, M.D., El Paso, Texas.

ANSWER.—Ptyalism, a rare complication of pregnancy, is related to hyperemesis and may occur alone but is generally associated with nausea and vomiting. It usually begins in the second month, ceases about the fifth month or at quickening, presents the same variations as vomiting and almost always ceases with parturition, but it has been known to continue for a few weeks

thereafter. It usually occurs only once but may appear in successive pregnancies and may be absent in one and recur in another. The flow varies in amount; excessive quantities have been reported—more than 1,920 cc. (2 quarts) a day. It usually lessens during the night but may continue unabated. The saliva is watery, tasteless, odorless, limpid and not acid; it has little ptyalin. The patient cannot swallow the saliva, and it nauseates her.

Ptyalism occurs more often in psychotic women. The diagnosis is not difficult, since mercurial salivation is attended by fetor and stomatitis. The treatment is similar to that of hyperemesis. The drain of this large amount of fluid, the absence of digestive powers and the loss of appetite can produce a condition of exhaustion, which resembles that produced by intractable vomiting and on rare occasions has been an indication for termination of pregnancy.

CYSTITIS FOLLOWING INTERCOURSE

To the Editor:—*A 41 year old white woman has had cystitis following intercourse many times during her married life of 10 years. Her general health previously and now has been excellent. The cystitis always follows intercourse by one to three days and never occurs other than at that time. Such attacks are easily terminated by the use of such agents as sulfonamides, aureomycin and mandelamine. Between attacks there are no abnormal urinary findings or symptoms. In 1941, a few months after marriage and following the second attack of cystitis, cystoscopic findings and retrograde pyelograms were normal. This patient has had two normal pregnancies with no complications. The genital tract is still normal. No urinary cultures have been made; however, a Gram stain of the urine sediment always shows a gram-negative, motile bacillus. The urine is always acid. What is the probable cause of so many recurrences, and what can be done to prevent them?*

James T. Hardy, M.D., Lake Worth, Fla.

Answer.—The first step in the solution of this patient's problem is a very careful examination of her genital tract. The inquiry states that results of the gynecological examination were negative, but it does not state how recent this was. The second step is cystoscopic examination, x-rays and intravenous pyelograms. The inquiry states that when these were made some years ago they were normal. The third point is the condition of her intestinal tract. Many patients with recurring attacks of so-called cystitis suffer from diseases of the gastrointestinal tract, such as cathartic colitis, spastic colitis, constipation, hemorrhoids and amebiasis. Therefore, this possibility must be ruled out. Last, but not least, is the significant statement that these attacks of cystitis follow intercourse. This is significant in view of other statements that everything else has been normal. If results of the genitourinary examination, the gynecological examination and the examination of the intestinal tract are all negative, there probably is a psychic element in this case, and she should consult a psychiatrist. It would be interesting to know whether sexual intercourse was painful. This clinical picture is not so uncommon.

LOW SPERM COUNT

To the Editor:—*What effects, if any, have cathode rays or any other rays present in certain departments of television companies? A healthy married man in his twenties, who is the father of a 4 year old child, born when he was a milk driver, now has a sperm count of only 6,000,000, with 50 per cent motile (high power field) and 8 to 10 pus cells. There is no history of gonorrhea. If these rays have no effect on spermatogenesis, what, if anything can be done to stimulate sperms? The wife is normal but has not become pregnant again. Workmen at this television company are of the opinion they are being exposed to a certain amount of x-rays given off by the television sets.*

A. E. A. Wanderer, M.D., Oak Park, Ill.

Answer.—It is hardly likely that the amount of cathode rays associated with television machines can penetrate deeply enough into the body to produce sterility. Therefore, some other cause for the low sperm count should be looked for. Generally, the attempts to increase a man's sperm count are not very successful. Nevertheless, every effort should be made to try to increase the procreative ability of the man. The diet should be discussed with the patient, because occasionally an improper diet may be responsible for impaired fertility. Vitamins, such as A and E, have been prescribed, but an impartial analysis reveals that these have not helped much to stimulate spermatogenesis. Other vitamins have not been tried out sufficiently.

In the discussion with this patient, the matters of excessive amount of work, fatigue, marital troubles and other anxieties should be discussed. Every effort should be made to overcome tiredness, overwork, excessive use of alcohol and sedentary habits. If the patient is overweight, an effort should be made to reduce the excess.

Endocrine products are being used extensively to increase spermatogenesis. The following substances are in use: (1) thyroid extract, (2) chorionic gonadotropins, (3) pituitary gonadotropins, (4) mare's serum gonadotropins and (5) androgens. In patients with hypothyroidism, thyroid extract is of distinct benefit; otherwise it usually is not of much help. The results with the gonadotropins are far from good, and some men will have foreign protein reactions from these gonadotropins. The male sex hormones are used, but they may be without benefit and may actually depress spermatogenesis.

It must be realized, therefore, that at present there is no satisfactory hormone treatment for defective spermatogenesis, but improvement can be obtained in some cases. There is as yet no specific sexual hormone with which to increase spermatogenesis.

SUBSTANCES USED TO STIMULATE FIBROUS TISSUE

To the Editor:—*The answer to the query on this subject in* The Journal *June 2, 1951 (page 509) makes no mention of work by Boyd, Saint, and me on the treatment of thoracic and abdominal aneurysms of syphilitic and arteriosclerotic origin by the periarterial injection of dicetyl phosphate dissolved in olive oil* (J. Indiana M. A. **42**:889 [Sept.] 1949). *I discussed this subject before the Central Surgical Association in February, 1951, at which time I reported that 0.9% dicetyl phosphate dissolved in olive oil produced an enormous amount of fibroplasia when injected around blood vessels in experimental animals. The olive oil must be heated to body temperature in order to keep the dicetyl phosphate in solution.*

We recently received a supply of sodium dicetyl phosphate from the Monsanto Chemical Company. This is a tribasic salt with two hydrophilic radicles, namely, sodium and phosphate. This leaves the dicetyl component as a suspension which is easily injectable and does not require heating. Moreover, the strength of the solution may be greatly increased over that used in olive oil. Dicetyl phosphate has been used in the treatment of aneurysm in four patients. The results have been very promising. As soon as a sufficient time for proper evaluation has elapsed, a report will be made of the clinical results.

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DIAPER RASH

To the Editor:—*In Queries and Minor Notes in* The Journal (**146**:*416* [*May 26*] *1951*), *a question is raised concerning ammonia dermatitis and its treatment with regard to possible agents given internally. This question was not directly answered, and no mention was made of oral preparations that could be used as prophylaxis or treatment. I would like to call attention to the article "Napkin Dermatitis Caused by Ammoniacal Urine: Treatment with Methionine" (Goldstein:* Arch. Pediat. **66**:*553-560* [*Dec.*] *1949). This is a practical paper, and a theoretical discussion of mode of action is presented.*

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